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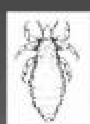


Regional
Disease Vector Ecology Profile

South Central Asia

Defense Pest Management Information Analysis Center
Armed Forces Pest Management Board
Forest Glen Section
Walter Reed Army Medical Center
Washington, DC 20307-5001

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I. PREFACE

Disease Vector Ecology Profiles (DVEPs) summarize unclassified literature on medically important arthropods, vertebrates and plants that may adversely affect troops in specific countries or regions around the world. Primary emphasis is on the epidemiology of arthropod-borne diseases and the bionomics and control of disease vectors. DVEPs have proved to be of significant value to commanders, medical planners, preventive medicine personnel, and particularly medical entomologists. These people use the information condensed in DVEPs to plan and implement prevention and control measures to protect deployed forces from disease, injury, and annoyance caused by vector and pest arthropods. Because the DVEP target audience is also responsible for protecting troops from venomous animals and poisonous plants, as well as zoonotic diseases for which arthropod vectors are unknown, limited material is provided on poisonous snakes, noxious plants, and diseases such as hantavirus.

In this document vector-borne diseases are presented in two groups: those with immediate impact on military operations (incubation period < 15 days) and those with delayed impact on military operations (incubation period > 15 days). For each disease, information is presented on military importance, transmission cycle, vector profiles, and vector surveillance and suppression.

Additional information on venomous vertebrates and noxious plants is available in the Armed Forces Medical Intelligence Center's (AFMIC) Medical, Environmental, Disease Intelligence, and Countermeasures (MEDIC) CD-ROM.

Contingency Operations Assistance: The Armed Forces Pest Management Board (AFPMB) is staffed with a Contingency Liaison Officer (CLO), who can help identify appropriate DoD personnel, equipment, and supplies necessary for vector surveillance and control during contingencies. Contact the CLO at Tel: (301) 295-8312, DSN: 295-8312, or Fax: (301) 295-7473.

Defense Pest Management Information Analysis Center (DPMIAC) Services: In addition to providing DVEPs, DPMIAC publishes Technical Information Bulletins (TIBs), Technical Information Memorandums (TIMs), and the Military Pest Management Handbook (MPMH). DPMIAC can provide online literature searches of databases on pest management, medical entomology, pest identification, pesticide toxicology, and other biomedical topics. Contact DPMIAC at Tel: (301) 295-7476, DSN: 295-7476, or Fax: (301) 295-7482. Additional hard copies or diskettes of this publication are also available.

Other Sources of Information: The epidemiologies of arthropod-borne diseases are constantly changing, especially in developing countries undergoing rapid growth, ecological change, and/or large migrations of refugee populations resulting from civil strife. In addition, diseases are underreported in developing countries with poor public health infrastructures. Therefore, DVEPs should be supplemented with the most current information on public health and geographic medicine. Users may obtain current disease risk assessments, additional information on parasitic and infectious diseases, and other aspects of medical intelligence from the Armed Forces Medical Intelligence Center (AFMIC), Fort Detrick, Frederick, MD 21701, Tel: (301) 619-7574, DSN: 343-7574.

Vector Risk Assessment Profiles (VECTRAPs) for most countries in the world can be obtained from the Navy Preventive Medicine Information System (NAPMIS) by contacting the Navy Environmental Health Center (NEHC) at Tel: (757) 762-5500, after hours at (757) 621-1967, DSN: 253-5500, or Fax: (757) 444-3672. Information is also available from the Defense Environmental Network and Information Exchange (DENIX). The homepage address is: <http://denix.army.mil/denix/denix.html>.

Specimen Identification Services: Specimen identification services and taxonomic keys can be obtained from the Walter Reed Biosystematics Unit (WRBU), Museum Support Center, MRC-534, Smithsonian Institution, Washington, DC 20560 USA; Tel: (301) 238-3165; Fax: (301) 238-3667; e-mail: <wrbu@wrbu.si.edu>; homepage: <http://wrbu.si.edu/>.

Emergency Procurement of Insect Repellents, Pesticides and Equipment: Deploying forces often need pesticides and equipment on short notice. The Defense Logistics Agency (DLA) has established the following Emergency Supply Operations Centers (ESOCs) to provide equipment and supplies to deploying forces:

> **For insect repellents, pesticides and pesticide application equipment:** Contact the Defense Supply Center Richmond ESOC at Tel: (804) 279-4865, DSN: 695-4865. The ESOC is staffed seven days a week/24 hours a day. Product Manager (804) 279-3995, DSN: 695-3995.
>
> **For personal protection equipment (bednets, headnets, etc.) and respirators:** Contact the Defense Supply Center Philadelphia ESOC Customer Assistance Branch at Tel: (215) 737-3041/3042/3043, DSN: 444-3041/3042/3043.

Every effort is made to ensure the accuracy of the information contained in DVEPs. Individuals having additional information, corrections, or suggestions, are encouraged to provide them to the Chief, DPMIAC, Armed Forces Pest Management Board, Forest Glen Section, Walter Reed Army Medical Center, Washington, DC 20307-5001, Tel: (301) 295-7476, DSN: 295-7476, or Fax: (301) 295-7482.

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II. EXECUTIVE SUMMARY

South Central Asia Profile

Geography. South Central Asia encompasses slightly more than 4.78 million sq km of land. From north to south, its diverse topography includes: 1) the world's highest mountains, the Himalayas, which include nine of the world's ten highest peaks; 2) the rugged ridges and narrow steep valleys of northern Pakistan, Nepal and India, drained by the Indus and Ganges Rivers; 3) the rich uplands of Kashmir and Uttar Pradesh; 4) coastal plains; and 5) low, flat, fertile deltas. It also includes the large subtropical island of Sri Lanka, with peaks up to 2,500 m elevation, and the 1,100 low coral islands in the atolls of the Maldives that straddle the equator and have a maximum elevation of only 2.4 m. Some of the most rugged and desolate areas in the world are found in Afghanistan, and in the Thar and Great Indian deserts, in southeastern Pakistan and northwestern India, respectively. South Central Asia is bordered on the north by Turkmenistan, Uzbekistan, Tajikistan, and China, on the east by Myanmar and the Bay of Bengal, on the south by the Indian Ocean, and on the west by Iran and the Arabian Sea. This area has been a major source of spices and tea and a focus of world trade for centuries. The economies of most countries in the region are based mainly on agricultural products, textiles, minerals, fishing, petroleum and tourism. Afghanistan, India, Pakistan, and Sri Lanka have all suffered fluctuating levels of bitter civil violence between different ethnic and religious groups, castes, and political factions throughout most of the past 20 years. India and Pakistan have teetered on the brink of border war since their founding as independent states. Nearly all of the countries in this region must import significant amounts of their energy resources, food, and raw materials.

Climate. The climate of South Central Asia varies widely, from arctic weather in northern mountainous areas, to temperate climates in higher and medium altitude valleys and plains, to tropical in coastal plains, river delta areas, and southern islands. Most of Afghanistan and the Thar and Great Indian Deserts are arid all year, very hot in summer yet very cold in winter. The climate in any particular country depends on its proximity to major mountains or large bodies of water, its elevation, and local topography. Coastal lowlands tend to have moderate, moist conditions year-round, while steep valleys at higher elevations tend to be cooler and drier, especially when they are on the lee side of prevailing weather patterns. In the southernmost countries, it can be very hot in mid-summer at lower elevations, especially on southeast-facing slopes. Wind and weather patterns tend to travel from southwest to northeast across this area during the year's first monsoon season, and northeast to southwest in the second monsoon season. Over most of South Central Asia, the timing, intensity, and duration of any given monsoon can greatly affect the available moisture, agricultural success, and overall economic success of a village or even a whole country for any particular year. Local wind direction, moisture content, and temperature extremes can be greatly modified by mountains, valley and ridge topography, or proximity to large bodies of water. Climatic extremes can be fatal. Humans face natural health hazards from high altitude, extreme cold, earthquakes and landslides in the rugged mountainous and steep hilly areas of Afghanistan, Bhutan, India, Nepal and Pakistan. In southern areas of the region, human populations are threatened by severe flooding from tides or monsoon storms, as well as heat injury in the hot, humid tropical and subtropical lowlands and seacoasts of Bangladesh, India, Maldives and Sri Lanka. Locally severe thunderstorms and tornadoes may occur anywhere from the mountains to the coasts of any of these countries. Extremely dry conditions are a threat to human life throughout much of Afghanistan, and the deserts of south-central Pakistan and northwestern India.

Population and Culture. The South Central Asian region has a population of approximately 1.34 billion people. The Indian Subcontinent was the site of development of some of the earliest civilizations and cultures, and is the historic and current center of at least 2 major religions, Hinduism and Buddhism. Most of the ethnic groups in this region have a long and proud history, passionate feelings for their heritage, and great awareness of their ancestry and cultural background. Over the centuries, the isolation imposed by extremely rugged terrain and limited transportation have allowed many small cultural units to develop independently. Consequently, the ethnic make-up, religious mix and culture of each country may vary greatly over short distances. Castes, families, and clans have very complex loyalties, and these may strongly influence people's daily lives. The religious make-up of the entire region is about 63% Hindus, 31% Muslims, 1.9% Christians, 1.7% Buddhists, 1.5% Sikhs, and about 1% Jains and others. There are

more than 30 different ethnic groups living in the region, and it is often hard to characterize the relative proportion of the total population that belongs to each of them. This great diversity, combined with difficult terrain and long cultural histories, has divided the people and caused frequent fighting between families, clans, castes, and states. Countries in this region are generally poor, underdeveloped, densely populated, and rely on agriculture as one of their main industries. Population densities range from a low of 39.9 persons per sq km in Afghanistan, to 1,101 persons per sq km in the densely populated Maldives. Illiteracy rates are high, varing from about 72% in Nepal to 7% in the Maldives. Urbanization rates vary from a low of about 6% for Bhutan to a high of 33% for Pakistan. Urbanization is occurring at extremely rapid rates in India and Pakistan. Most of the large urban areas contain extensive, overcrowded slums and shantytowns in which epidemics of infectious and arthropod-borne disease are common.

Sanitation and Living Conditions. Sanitation and living conditions throughout this region are marginal to barely adequate by Western standards. Septic and sewer systems are found in a few of the larger cities, but these are often inadequate, old, and poorly maintained. Water treatment and distribution systems are few, limited (many serve far less than half of their city), and poorly maintained, and frequent cross-connections result in contamination by human or animal fecal matter or industrial wastes. Hepatitis E and many gastrointestinal infections are commonly acquired by consuming sewage-contaminated drinking water. Waste disposal is indiscriminate in many areas, especially urban slums, where waste and refuse are sometimes piled in ditches, roadsides, or alongside buildings. Night soil is commonly used as fertilizer for crops. Food sanitation is poor. Rodents, flies and mosquitoes are abundant in and around human dwellings in many areas of the region. Housing shortages in many cities have been made worse by an influx of refugees, and several families may share a small building. Areas with sanitary conditions degraded by civil war have rapidly increasing rodent populations. The potential for rodent-borne, water-borne, food-borne and insect vector-borne diseases under such crowded, unsanitary conditions is high. Many cities, streams and rivers, and even some extensive agricultural areas, especially along streams and river deltas, have been contaminated by heavy metals and toxic chemicals from industrial activity, such as textile production, chemical manufacturing, and petroleum refining. Very little effort has been made to reduce or prevent industrial pollution. Countries with serious environmental problems as a result of industrial pollution include India and Pakistan.

DIARRHEAL DISEASE

Gastrointestinal infections are highly endemic throughout South Central Asia and are the principal disease threats to military personnel deployed to the region. Bacillary dysentery has had a profound impact on military operations throughout history.

Fecal-oral transmission from person to person is common, but most infections are acquired from the consumption of contaminated food, water or ice. Filth flies can be important in the mechanical transmission of pathogens to food, food preparation surfaces and utensils. Fly populations sometimes reach very high levels during the summer in areas with poor sanitation. Strict sanitation and fly control can significantly reduce the risk of gastrointestinal infections. Cockroaches have also been shown to mechanically transmit gastrointestinal pathogens.

Bacteria and viruses causing diarrheal disease include: *Staphylococcus aureus*, *Clostridium perfringens*, *Bacillus cereus*, *Vibrio parahaemolyticus*, numerous serotypes of *Salmonella*, *Shigella* spp., *Campylobacter*, pathogenic strains of *Escherichia coli*, hepatitis A and E, rotaviruses, and other viral species. Infection with pathogenic protozoa, such as *Entamoeba histolytica*, *Giardia lamblia* and *Cryptosporidium* spp., is common, though bacterial pathogens account for most cases of diarrheal disease. Onset of symptoms is usually acute and may result in subclinical infections or severe gastroenteritis. *Shigella* infections can produce significant mortality even in hospitalized cases. Typhoid and paratyphoid fevers are also highly endemic and were a major cause of illness among Soviet troops serving in Afghanistan during the 1980s.
Resistance of enteric pathogens to commonly used antibiotics can complicate treatment. Such resistance is very common in many parts of South Central Asia, and bacterial populations with resistance to multiple antibiotics have been reported.

MOSQUITO-BORNE DISEASE*

Malaria is widely distributed in all countries of the region except the Maldives, including urban as well as rural areas. It is the most significant arthropod-borne disease threat to military forces operating in South Central Asia. Vivax malaria predominates in Afghanistan, Bhutan, Nepal, Pakistan and Sri Lanka, while falciparum malaria causes most cases in Bangladesh and India. Chloroquine-resistant falciparum malaria has been reported from every country in the region. Resistance to Fansidar™ has been reported from Bangladesh, Bhutan, India and Pakistan. More ominous are reports from India of strains of *Plasmodium vivax* resistant to chloroquine and primaquine. At least 20 species of *Anopheles* mosquitoes transmit malaria in the region. Insecticide resistance is widespread.

Annual epidemics of **dengue** occur throughout South Central Asia except for Afghanistan and Nepal. Populations of the primary vector, *Aedes aegypti*, have greatly increased as a result of rapid and uncontrolled urbanization. *Aedes albopictus* is an important vector in rural areas of the region. **Dengue hemorrhagic fever** and **dengue shock syndrome** have been reported from several areas of India, Karachi in Pakistan, and recently from Bangladesh. This debilitating disease would be a significant threat to military forces operating in the region.

Japanese encephalitis (JE) is a serious neurological disease that causes high morbidity and mortality throughout Asia. Periodic outbreaks of JE have occurred in every country of South Central Asia except the Maldives. The expansion of irrigation and rice growing in many areas has greatly increased populations of the primary mosquito vector, *Culex tritaeniorhynchus*, as well as other *Culex* vectors. Consequently, the incidence of JE has increased. An effective vaccine will limit the detrimental effects of this disease in military personnel operating in endemic areas.

Sporadic but irregular outbreaks of **chikungunya fever** occur in Bangladesh, India, Nepal and Sri Lanka. This disease is transmitted by *Ae. aegypti*. Like dengue, it is debilitating and can incapacitate large numbers of people in a short period. Due to its limited prevalence in South Central Asia, chikungunya fever is less of a threat than dengue.

West Nile fever virus and **Sindbis virus** are circulating in India and Pakistan. Their enzootic status is unclear, but they represent little threat to military personnel due to the mild nature of the illnesses they cause and the apparently low prevalence of both viruses in South Central Asia.

Bancroftian filariasis caused by ***Wuchereria bancrofti*** is a major public health problem in India, where there is a minimum of 40 million cases. It also occurs in Bangladesh, Nepal, Pakistan and Sri Lanka. The primary vector, *Culex quinquefasciatus*, has become increasingly abundant as breeding sites have proliferated in urban slums and shantytowns with poor sanitation. The incidence of Bancroftian filariasis has been rapidly increasing in rural areas. **Brugian filariasis**, caused by ***Brugia malayi***, occurs at low levels in Pakistan but is prevalent in southern India, especially in Kerala State. It is transmitted primarily by several species of night-biting *Mansonia* mosquitoes. Serious medical complications result only from chronic infection, however the first infection with these filarial worms can produce acute illness in nonimmunes.

TICK-BORNE DISEASE*

Crimean-Congo hemorrhagic fever infects domestic animals in nearly every country in the region and is widely distributed in discrete foci in agricultural and rural areas. Human cases are sporadic. The most recent outbreaks have occurred in Afghanistan. The disease can be contracted by the bite of infected *Hyalomma* ticks, but most human cases result from exposure to secretions or blood from infected animals or humans. Medical workers treating patients are at high risk of becoming infected. Clinical symptoms can be severe, with mortality rates up to 50%.

Boutonneuse fever (also termed Indian tick typhus and Siberian tick typhus) transmitted by the brown dog tick, *Rhipicephalus sanguineus*, and other ixodid ticks is focally distributed, primarily in India, Nepal, Pakistan and Sri Lanka. The risk of infection is elevated in cities and villages with high populations of tick-infested dogs.

About 400 to 500 human cases of **Kyasanur Forest disease** have occurred annually in India since the disease first appeared in 1957. All human cases have been associated with forest exposure in several districts of Karnataka State. Disease reservoirs include rodents, shrews and monkeys. The primary vectors are *Haemaphysalis* ticks, particularly *H. spinigera*. The disease poses little military threat due to its limited distribution.

Q fever is an acute, febrile rickettsial disease contracted primarily by inhalation of airborne pathogens or contact with secretions of infected domestic animals. Transmission by ticks to humans is possible but rarely, if ever, occurs. Serological surveys indicate that Q fever is widespread throughout South Central Asia and infects a wide variety of wild and domestic animals, especially sheep and goats. Military personnel should avoid exposure to sheep, goats, cattle and other domestic animals and should not sleep or rest in animal shelters.

Sporadic cases of **tick-borne relapsing fever**, caused by *Borrelia recurrentis*, are also reported throughout the region. The disease is enzootic in rocky, rural areas where livestock are tended and vector soft ticks, *Ornithodoros* spp., are found.

MITE-BORNE DISEASE*

Scrub typhus, caused by the rickettsia *Orientia tsutsugamushi*, is focally distributed throughout the region from coastal lowlands to over 3,000 m elevation in the Himalayan Mountains. The disease is transmitted by chigger mites of the genus *Leptotrombidium* that are primarily associated with rodents of the genus *Rattus*. Scrub typhus is prevalent in disturbed habitat characterized by secondary scrub vegetation and grasses. During World War II, scrub typhus was one of the leading causes of morbidity in military personnel in the Asia-Pacific area. Although the incidence of human infection in South Central Asia is unknown, the disease is a significant threat to military forces in the field.

LOUSE-BORNE DISEASE*

Epidemic typhus may still be endemic in poor people living in the Himalayas. Declining sanitary and living conditions caused by civil war in many areas of Afghanistan have increased the incidence of head and body lice. Cases of epidemic typhus were reported in Afghan refugees living in camps along the Pakistani border. In addition, 90,000 refugees fled civil unrest in Bhutan during the 1990s and live in seven Nepalese refugee camps administered by the United Nations. The conditions faced by refugees and displaced persons, whether in collective centers or living independently in the community, are such that the likelihood of louse-borne disease is high. Incidence of body lice in many areas of South Central Asia is high.

Sporadic cases of **louse-borne relapsing fever** have also been reported from the region, and, like epidemic typhus, it is a winter disease.

FLEA-BORNE DISEASE*

Murine typhus is a rickettsial disease similar to louse-borne typhus but milder. It is enzootic throughout the region in domestic rats and mice and possibly other small mammals. Infected rat fleas, usually *Xenopsylla cheopis*, defecate infective rickettsiae while sucking blood. Airborne infections can occur. Sporadic human cases have been reported throughout South Central Asia.

A large outbreak of **plague** involving nearly 6,000 suspected cases occurred in the Indian states of Maharashtra and Gujurat in 1994. These were the first cases of human plague reported from India since 1966. Cases of pneumonic plague occurred in the city of Surat, and the epidemic spread mass panic in the country. The current enzootic status of plague in South Central Asia is poorly known.

SAND FLY-BORNE DISEASE*

Foci of **sand fly fever** occur throughout the coastal areas of South Central Asia. It is also found at lower altitudes of mountainous areas along river valleys of Afghanistan, Pakistan, northern India and Nepal. During World War II, this disease caused significant morbidity among Allied forces in the China-Burma-India theater as well as other areas. Although there are no fatalities and victims are sick usually for no more than one or two weeks, the disease is potentially of great military importance, since large numbers of

personnel can be incapacitated in a short period. The highest risk of transmission exists in villages and periurban areas. Local populations are generally immune as a result of childhood infection. Both the Naples and Sicilian viruses circulate in endemic areas of South Central Asia. Risk of infection is highest between April and October, when the primary sand fly vector, *Phlebotomus papatasi*, is most active. *Phlebotomus argentipes* is an important secondary vector in the region. Humans are the reservoir of this debilitating disease, although small rodents are suspected reservoirs.

Cutaneous leishmaniasis is moderately or highly endemic regionally. Two species of *Leishmania* cause skin lesions in the region. The less severe and rurally distributed *Leishmania major* is a parasite of desert and dry jungle rodents, especially gerbils such as *Rhombomys*, which may be 60 to 100 percent infected. Cutaneous leishmaniasis caused by *Le. major* and called "rural" or "wet" cutaneous leishmaniasis or zoonotic cutaneous leishmaniasis is present in small villages and rural areas of South Central Asia. The proven sand fly vector of *Le. major* in South Central Asia is *Phlebotomus papatasi*.

The second type of **cutaneous leishmaniasis** is caused by *Leishmania tropica* and called "urban" or "dry" cutaneous leishmaniasis or anthoponotic cutaneous leishmaniasis. *Le. tropica* is usually a parasite of man in urban environments and is transmitted primarily by the sand fly *Phlebotomus sergenti* in Afghanistan, India, and Pakistan. *Phlebotomus sergenti* is very tolerant of the high summer temperatures and cold winter temperatures found in the upper mountain passes and plateaus of South Central Asian countries. Man is the principal reservoir, but dogs have been found naturally infected. Cases of visceralizing *Le. tropica* were found among American soldiers stationed in Saudi Arabia during the Persian Gulf War and have been reported from India and Pakistan.

Visceral leishmaniasis (VL), caused by two parasites, *Leishmania donovani* and *Le. infantum*, is a less prevalent but more severe systemic disease. VL often reaches epidemic proportions in both urban and rural areas. VL caused by *Le. donovani* is spread by the anthroponotic sand fly vector *Phlebotomus argentipes* in southern costal India and Bangladesh. Man is believed to be the reservoir of *Le. donovani* in South Central Asia. In recent years, VL caused by *Le. infantum* has appeared in Pakistan and Afghanistan as well as northern India, generally as foci in rural areas. Suspected vectors of *Le. infantum* in western Central Asia are *P. chinensis*, *P. caucasicus*, *P. kandelakii s. l.*, *P. longiductus* and *P. major*, which are found in more mountainous areas. The most common reservoirs of *Le. infantum* are believed to be domestic dogs and wild canines, primarily foxes. Transmission of both types of VL occurs during the warmer months of April through October, coinciding with the activity of vector sand flies. Phlebotomine sand flies bite from dusk to dawn but may feed during the day if hosts enter their resting habitat. The distribution of sand flies and the diseases they carry is often very focal because of larval requirements for humid organic microhabitats protected from sunlight and limited adult flight capabilities. The incidence of leishmaniasis has been rapidly increasing in some areas as a result of the cessation of spraying of residual insecticides for malaria control.

RODENT-BORNE DISEASE

Hantaviral diseases are an emerging public and military health threat. Field rodents are reservoirs for several closely related viruses that can be transmitted to humans exposed to airborne pathogens from dried rodent excreta. Serological evidence of hantaviral infection has been detected in humans or wild animals in India, Nepal and Sri Lanka. At least 3 serotypes of hantavirus that cause clinical disease in humans are present in the region. A severe form of the disease referred to as hemorrhagic fever with renal syndrome is caused by Hantaan virus and is associated with field mice in open field or unforested habitats. Death rates often exceed 10%. A milder disease, caused by Puumala virus, is associated with voles in woodland or forest-steppe habitats. The Seoul serotype infects *Rattus norvegicus*, and risk of infection occurs when living or working in dusty rat-infested buildings. Thottapalayam virus has been isolated from *Suncus* shrews in India, but its role in human disease is poorly known. Very few cases of human disease have been reported from the region, and little is known about the epidemiology of hantaviruses in South Central Asia.

Leptospirosis should be considered enzootic in most countries of South Central Asia. The spirochete is transmitted when skin or mucous membranes are contacted by water contaminated with urine of infected domestic and wild animals, especially rats. Military personnel would be at high risk of infection from this

disease. Troops should never handle rodents and should not sleep or rest near rodent burrows or swim or bathe in stagnant pools or sluggish streams.

SNAIL-BORNE DISEASE

Only two small, insignificant foci of **schistosomiasis** occur in South Central Asia. Intestinal schistosomiasis, caused by *Schistosoma mansoni*, has been detected in inhabitants of Dhanusha District in southern Nepal. A focus of urinary schistosomiasis, caused by *S. haematobium*, has been known since 1952 in Gimvi village, Ratnagiri District, Maharashtra State, India.

CONJUNCTIVITIS

Bacterial and viral **conjunctivitis** is very common in South Central Asia and has epidemic potential. An outbreak of acute hemorrhagic conjunctivitis occurred in Delhi, India, during August and September of 1996. The etiologic agent was confirmed as enterovirus type 70. After nearly a decade, this virus is re-emerging as a cause of acute hemorrhagic conjunctivitis. Transmission is normally through contact with secretions of infected persons or contaminated articles. Although blinding **trachoma**, caused by the bacterium *Chlamydia trachomatis*, has become less prevalent in India, it is still the leading cause of blindness in Nepal. Non-blinding trachoma continues to be a serious public health problem throughout South Central Asia. During surveys conducted in the mid 1990s, active trachoma was found in 18% of school children under 15 years of age in Delhi, in 8.5% of children under 10 years of age from the western parts of the North Indian State of Uttar Pradesh, in 23.6% of the preschool children in Sarlahi District, Nepal, and in 3.7% of Afghan refugee children under 10 years of age in Pakistan. Eye gnats and flies can mechanically transmit these pathogens. Several species of eye-frequenting moths, primarily in the family Noctuidae, are known from South Central Asia. These moths feed on the lacrimal secretions of wild or domestic animals as well as humans. They may also play a role in the transmission of ocular pathogens.

VENOMOUS ANIMALS

Forty-seven species of poisonous terrestrial snakes and 24 species of venomous sea snakes representing 5 families are found in the South Central Asian region. Some, such as the king cobra that can reach 18 feet in length, can be very intimidating and possess lethal venoms. Snakebite is a serious risk in the region. Military personnel should be thoroughly briefed on the risk and prevention of snakebite, as well as the steps to take immediately after snakebite. Effective antivenoms are available. Scorpions, centipedes and widow spiders (*Latrodectus* spp.) are common in many parts of the region. A number of scorpion species, such as *Mesobuthus eupeus*, *M. hendersoni*, *M. pachyurus*, *M. tamulus* and *Ondotrobuthus ondoturus*, are notably toxic and may cause a variety of painful symptoms in adults, including neurological involvement. Death is usually limited to small children and the elderly. Scorpion stings rarely require hospitalization, although envenomization by widow spiders can be life threatening. Troops should be warned not to tease or play with snakes and scorpions.

*** A properly worn Battle Dress Uniform (BDU) impregnated with permethrin, combined with use of extended duration DEET on exposed skin, has been demonstrated to provide nearly 100% protection against most blood-sucking arthropods. This dual use of highly effective repellents on the skin and clothing is termed the "DoD arthropod repellent system." It is the most important single method of protecting individuals against arthropod-borne diseases. Permethrin can also be applied to bednets, tents and screens to help prevent disease transmission by insects. The proper use of repellents is discussed in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance.**

VECTOR-BORNE DISEASES IN SOUTH CENTRAL ASIA (+ = present; ? = uncertain).

Diseases	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
malaria	+	+	+	+		+	+	+
Japanese encephalitis	+	+	+	+	?	+	+	+
dengue		+	+	+	+	?	+	+
chikungunya fever		+		+		?	+	+
West Nile fever				+			+	
Sindbis virus				+				
sand fly fever	+	+	?	+	?		+	
plague	?			+		?		
murine typhus	+	+	+	+	?	+	+	+
Kyasanur Forest disease				+				
boutonneuse fever				+		+	+	+
tick-borne relapsing fever	+			+			+	
Crimean-Congo hemorrhagic fever	+			+	+	+	+	
Q fever	+	?		+			+	+
scrub typhus	+	+		+	?	+	+	+
louse-borne typhus	+					?	?	
louse-borne relapsing fever	?	?	?	?		?	?	
leptospirosis	+	+	?	+	?	+	+	+
hantaviral disease				+		?		+

VECTOR-BORNE DISEASES IN SOUTH CENTRAL ASIA (+ = present; ? = uncertain).

Diseases	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
visceral leishmaniasis	+	+	+	+		+	+	?
cutaneous leishmaniasis	+	+		+			+	+
filariasis		+	?	+		+	+	+
schistosomiasis				+		+		

III. Map of South Central Asia.

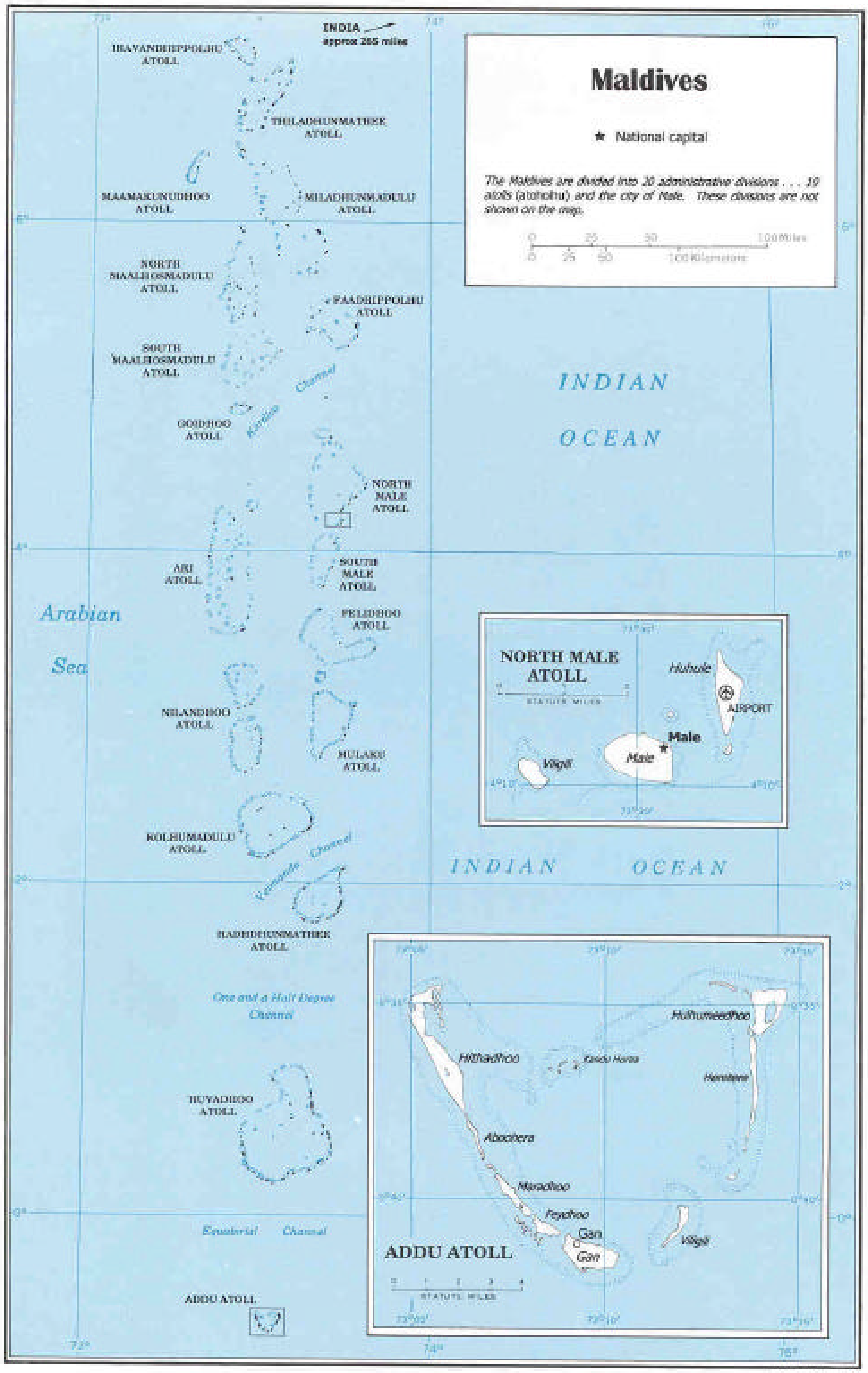
A. Map of Afghanistan.

B. Map of Bangladesh.

C. Map of Bhutan.

D. Map of India.

E. Map of Maldives.



F. Map of Nepal.

G. Map of Pakistan.

H. Map of Sri Lanka.

IV. Country Profiles.

A. Afghanistan.

1. Geography. Afghanistan is landlocked, bordered by China, Iran, Pakistan, Tajikistan, Turkmenistan and Uzbekistan. It has 647,500 sq km of land area, slightly smaller than Texas, and can be divided into three geographic regions: (1) The Northern Plains, covering about 16% of Afghanistan, consist of mountainous plateaus and rolling hills, averaging 600 m in elevation. (2) The Central Highlands, part of the Himalayan Chain, cross the center of Afghanistan from the northeast to the southwest, and cover almost two-thirds of the country. Peak elevations occur in the northeast and reach 7,485 m at Nowshak. The mountains of Afghanistan form a natural divide, separating the Indian Subcontinent from Central Asia to the northwest and Iran to the west. (3) The Southwestern Plateau is southwesterly sloping and arid, with elevations reaching 6,000 m in the east, dropping to 258 m at Amu Darya in the west. Earthquakes cause severe damage in the Hindu Kush mountains that occupy the northeastem one-third of the country. The arid climate, weather extremes, rugged terrain, sparse vegetation, deforestation, and overgrazing are causing rapidly expanding desert conditions. Only 12% of the land is arable, and 46% is in permanent pasture. Most of the country is treeless. In the low valleys, agriculture is intensively pursued and native poplar trees provide wood for rough houses. More than 70% of the land is not accessible because of the rugged terrain, impassable roads, and land mines remaining after years of fighting. In 1996, Afghanistan had an estimated 21,000 km of roads, of which only 2,793 (13%) were paved, and there were only 25 km of railroads. Afghanistan has an environment hostile to human comfort and even survival. Earthquakes, extremes of heat and cold, high altitude, and blowing sand are threats to human health. Unexploded mines, artillery shells, and grenades scattered or buried over much of the countryside maim and kill hundreds annually.

2. Climate. Afghanistan's climate is arid to semi-arid, with extremely dry, hot summers, but with cool nights, and cold winters with mountain blizzards. It has the widest annual temperature range of any country in the world, from – 50^{o}C to + 47^{o}C. There are daily and seasonal temperature extremes as well. In the west, mean daily maximum temperatures in summer (April through September) and winter (October through March) generally are 11^{o}C warmer than those in Kabul; winter highs and lows are 38^{o}C and -19^{o}C. Countrywide, the summer high temperature is 48^{o}C in the west and the low temperature is 20^{o}C in Kabul. The rainy season lasts from October through April, providing moderate rain and snow in the mountains and scanty rain on the plain. Despite limited amounts of rainfall, periodic heavy rains combined with melting snow have caused serious local flooding. Generally, no area receives more than 380 mm of precipitation annually. The country also has the "wind of 120 days," which blows from June through September at velocities reaching 177 kph and causes severe dust storms.

More than 60% of the nation's food harvest was lost due to a severe drought during 1999. The drought has continued into 2001 worsening the already desperate living conditions. Over 1 million people are at risk of famine. So bad has the situation become that in the past year over 700,000 Afghans have abandoned their homes in an attempt to cross the border into Pakistan or Iran. Countless thousands live under miserable conditions in refugee camps in Talacon of eastern Afghanistan and near Herat in western Afghanistan. More than 1 million Afghan refugees remain in Pakistan from the last big exodus during the war with the Soviet Union. The Paskistani government has virtually closed the border to prevent influx of additional refugees. Infectious disease is common in these refugee camps characterized by overcrowding, poor sanitation and malnutrition.

Kabul (elevation 1,791 m)												
Mean Daily Temperatures (^oC)												
MONTH	**J**	**F**	**M**	**A**	**M**	**J**	**J**	**A**	**S**	**O**	**N**	**D**
MAXIMUM	3	5	11	19	24	29	31	31	27	21	14	7
MINIMUM	- 5	- 4	3	9	12	16	20	19	14	7	1	- 3
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	30	36	94	102	20	5	3	3	0	15	20	10

3. Population and Culture. Afghanistan is an economically poor nation, whose medical, educational, and industrial infrastructure has been nearly destroyed by 2 decades of international and civil war. An estimated 2 million people were killed and 6 million refugees fled to Iran and Pakistan from 1979 to 1989. Roughly two thirds of those have since returned to Afghanistan. Of the nearly 1 million internally displaced refugees throughout Afghanistan, approximately 500,000 are from Kabul and reside in refugee camps near Jalalabad. Economic and public health problems are not being addressed, since the country has no functional national government and is essentially divided along ethnic lines. The complex politics and power struggles have left the Taliban in control of the capital and about 90% of the country. They have declared that they are the legitimate government of the nation. However, the U.N. has deferred a decision on credentials, and the Organization of the Islamic Conference has left the Afghan seat vacant until negotiations among the warring parties can resolve key issues. The remaining one-third of the country, in the ethnically diverse north, is under the control of the Taliban's opponents. Afghanistan's population is mostly rural and illiterate. Family, tribal, clan, and cultural ties and alliances are very strong. Most of the people are poor, often nomadic, and livelihoods are closely associated with the land. Ethnic groups are fiercely independent and treasure their heritage. The people identify themselves mainly on the basis of language. Ethnic make-up of the population is about 38% Pashtun (Afghans), 25% Tajik, 19% Hazara, and 12% Uzbeks, Turkmens and others. Religious make-up of the population is roughly 84% Sunni Muslim, 15% Shi'a Muslim, and 1% others. Total population 25.8 million (1999 estimate) with an average density of 39.9 persons per sq km; 18% urbanized; literacy rate 32%, although the female illiteracy rate is estimated at 85%. More than half of the total urban population lives in the capital, Kabul.

4. Sanitation and Living Conditions. Sources of water include rivers, streams, melted snow, irrigation ditches, gutters, and open wells. Seasonal water shortages are common. Approximately one-third of Kabul's population receives water from piped water systems, with the rest obtaining water from public or private wells. Water treatment and distribution systems are unreliable because of the war-torn infrastructure. Contamination of potable water supplies is a serious public health problem. Several international non-governmental agencies are involved in improving water services in Afghanistan. Sanitary conditions are poor throughout the country and are often worse in urban areas because of continuing civil strife. Sanitary sewage disposal is ineffective. In Kabul, septic tank systems are in disrepair, and indiscriminate defecation is common. Drainage canals carry human waste and other refuse directly to the Kabul River and open spaces in the city. These canals often overflow in the streets during spring rains. Poor personal hygiene and inferior food sanitation is the norm countrywide. Consequently, food-borne and water-borne diseases are common throughout the country, including cholera, shigellosis, typhoid fever, viral hepatitis and intestinal helminthic infections.

B. Bangladesh.

1. Geography. Bangladesh, approximately the size of Wisconsin, has a total land area of about 133,900 sq km. It is bordered on the north and west by India, on the southeast by Myanmar, and on the south by the Indian Ocean. Bangladesh can be divided into 2 major geographic regions: (1) The Bengal Plain (Lower Indo-Gangetic Plain), the largest delta in the world, covers most of the country. Its coastal marshes, mangrove forests, exceptionally fertile soil, and a vast estuary offer little protection to inhabitants from catastrophic typhoons that regularly occur. (2) The Chittagong Hill Tract, with a maximum elevation of

1,230 m at Keodradong, is a minor hill system in southeastern Bangladesh that constitutes one-sixth of the country. Severe deforestation has occurred in these highland areas. There are significant energy resources that are still undeveloped. A 1993 estimate listed 73% of the land as arable and 15 % as forest or woodland. Agriculture is the country's most important industry, and rice is the main crop. Intermittent water shortages occur in the northern and central parts of the country due to falling water tables. Soil degradation and deforestation are serious environmental problems. Severe storms and flooding are annual threats, especially during the monsoon season, and cause the loss of many lives every year. The 1988 monsoon floods submerged 75% of the country. Many people are landless and are thus forced to live on and cultivate flood-prone areas. Transportation and infrastructure improvements are very slow due to governmental inefficiencies and delays. There are 3,000 miles of navigable rivers but only about 6,500 miles of surfaced roads. The marshy ground and many rivers in the delta make road building difficult and costly.

2. Climate. Three seasons influence the tropical climate of Bangladesh. A dry, sunny, cooler season (October through February) has a mean daily maximum temperature of 28 ^oC. The hot season (March through June) brings high humidity, mean daily maximum temperatures of 32 ^oC, violent thunderstorms and tornadoes. July through September marks the monsoon season, with mean daily maximum temperatures of 31 ^oC, high humidity, and frequent rains that provide 80% of the country's annual precipitation (1,270 to 2,540 mm). Severe cyclones occur most frequently from September through October. A devastating cyclone in 1991 killed over 130,000 people. Cholera epidemics frequently follow severe flooding.

Dhaka (elevation 8 m)

Mean Daily Temperatures (^oC)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	25	27	32	35	33	32	31	31	31	31	28	26
MINIMUM	12	12	16	22	25	26	26	26	26	23	17	12

Monthly Precipitation (liquid equivalent)

MEAN (mm)	18	30	58	104	193	320	437	305	254	170	28	3

3. Population and Culture. Bangladesh remains one of the world's poorest and most densely-populated countries. Eighty-five percent of the population lives in more than 85,000 poorly defined villages. The overall population density exceeds 949 residents per sq km. The country's population is expected to exceed 240 million by the year 2030. Over 500,000 Muslims have illegally immigrated across India's border to the state of Assam since 1971. The competition with native Hindus for scarce jobs and land has resulted in political violence in Assam and tensions between India and Bangladesh. Ethnically, Bangladesh is roughly 98% Bengali. Biharis number about 250,000, and other tribal groups total less than 1 million. The religious make-up is about 88.3% Muslim, 10.5% Hindu, and 1.2% others. Family and clan ties are strong, and cultural heritage strongly influences most people's daily lives. Total population 127.1 million; 17% urbanized; literacy rate 38%.

4. Sanitation and Living Conditions. Rural and urban residents obtain water from wells, canals, ditches, ponds, streams and rivers. More than two million hand tubewells exist throughout the country. Coastal ground water sources are subject to saline intrusion. Water shortages exist in the northwest in part due to the Indian diversion of water from the Ganges River at the Farakka Barrage for the benefit of Calcutta. Shortages have also occurred in southwestern Bangladesh. Despite national and international efforts to develop adequate water supplies, water treatment and distribution in Bangladesh are extremely limited. Commonly, more than 1,000 inhabitants get water from a single, contaminated well. Less than one-half of residents in Dhaka have access to piped water, which is obtained from either deep wells or rivers and is treated at filtration plants. However, the distribution system is inadequate and water is usually

contaminated during distribution. Illegal taps into the system and back siphonage result in further contamination. Street hydrants are commonly used to supply water in the poorer sections of cities. Sanitary conditions, including food sanitation, are far below Western standards. Municipal sewage systems are available to less than 6% of the population. Refuse accumulates in the streets, attracting flies, rodents and feral dogs. In rural areas, night soil is routinely used as fertilizer. Water, air, and soil pollution are widespread. Untreated sewage and industrial wastes are discharged into surface waters that are also used for drinking, cooking, and washing. Indiscriminately used pesticides have contaminated many surface and ground water sources. Waterways often overflow during floods, causing epidemics of water-borne diseases. Arsenic contamination of ground water has been detected in several districts in central and southern Bangladesh and along the western border with India. Well water is frequently contaminated, and the risk of arsenic poisoning is considerable, shifting more reliance to surface waters. Commonly used slash-and-burn agricultural methods cause deforestation, soil erosion, clogging of waterways with silt, and air pollution.

C. Bhutan.

1. Geography. Geographically, Bhutan resembles Nepal. The country is landlocked and bordered by China and India. Bhutan's total land area of 47,000 sq km, slightly smaller than the state of Mississippi, can be divided into 3 geographic regions: (1) In the north, the Great Himalayas dominate the terrain, with peaks rising to more than 7,300 m above sea level (the highest point is Kula Kangri, at 7553 m), and high mountain valleys between 3,700 and 5,500 m above sea level. This area forms the northern border with China. (2) The Lesser Himalayas consist of southward spurs from the Great Himalayas, with peak elevations ranging from 1,500 to 4,270 m. (3) The narrow (13 to 16 km wide) Duars Plain, located at the base of the Lesser Himalayas, forms the southern border with India. The country's lowest point is at Drangme Chhu, at 97 m elevation. Bhutan translates as "Land of the Thunder Dragon," probably a reference to the violent storms that come down from the Himalayas. Landslides can be frequent and severe, especially during the rainy season. Bhutan has several rivers that originate in the Great Himalayas in the north and flow southward through intensively cultivated valleys. The rivers all flow into India, emptying into the Brahmaputra River. Roughly 66% of the land is forests and woodlands, and only 2% is arable. Much of the country is rugged or snow-covered, making access and transportation difficult. Bhutan's economy, one of the smallest and least developed in the world, is based on agriculture and forestry, which employ more than 90% of the population. The main trading partner is India. There is much potential for developing hydroelectric power.

2. Climate. Climatic conditions in Bhutan vary by geographic region. (1) In the northern mountainous region, winters (September through May) are long and severe; temperatures rarely rise above 0 ^{o}C. The region of the Great Himalayas is so cold that glaciers are present year-round. The short summers (June through August) have a mean daily temperature of 20 ^{o}C, although permanent frost prevails at higher elevations (5,000 m and above). Summer brings most of the region's 500 mm of annual precipitation. (2) Below 3,650 m elevation, the Lesser Himalayas have cool winters, with a mean daily temperature of 1 ^{o}C, and warm summers, with a mean daily temperature of 20 ^{o}C. Temperatures drop rapidly above 3,650 m elevation. Throughout the Lesser Himalayas, the average annual rainfall is approximately 1,250 mm. The south slopes of the Himalayas receive the most rain and are covered with thick forests. (3) The Duars Plain has a subtropical climate with high humidity and heavy rainfall. The rainy season, which usually lasts from May through December, brings most of the region's 5,685 mm of annual precipitation. Torrential rains occur in the southern fringe of the country. Mean daily temperatures are 5 ^{o}C during the cooler months and 25 ^{o}C during warmer months.

Thimphu (elevation 2,426 m)

Mean Daily Temperatures (^oC)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	18	19	25	28	30	29	28	28	28	26	23	19
MINIMUM	1	3	7	11	16	19	20	20	18	13	7	2
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	15	41	23	58	122	246	373	345	155	38	8	3

3. Population and Culture. Bhutan is a monarchy whose population is overwhelmingly rural, semi-nomadic and very poor. Many still herd yaks for a living. Modernization of the nation's infrastructure and economy, begun in 1960, has been intentionally limited by the government. Ethnic make-up is 50% Bhote, 35% Nepalese, and 15% other indigenous or migrant tribes. Religious preferences are 75% Lamaistic Buddhism and 25% Indian- or Nepalese-influenced Hinduism. Buddhism is the official state religion. Family and tribal heritage and culture are unique and strongly influence the daily lives of most people. The rugged terrain and limited roads leave valleys and remote villages isolated for much of the year. One small national airline provides the only regular air transportation to and from Bhutan. Total population 1.95 million; 13% urbanized; literacy rate 42%. Average population density is about 41 persons per sq km.

4. Sanitation and Living Conditions. Water sources include wells, springs, and rivers. Seasonal water shortages exist, especially at higher elevations and in isolated valleys and villages. In Thimphu, the capital, water is chlorinated during treatment; however, water may be contaminated at the tap because of back siphonage and defects in the distribution system. Most urban and almost all rural inhabitants obtain water directly from rivers and springs or neighborhood wells and standpipes. Several international non-governmental agencies are involved in improving water services in Bhutan. Sanitary conditions are poor throughout the country. Thimphu has a sewage system, but only part of the city has service. Bhutan has no solid waste collection system. Contamination of water and soil with untreated human and animal waste is a severe problem in much of Bhutan. Consequently, food-borne and water-borne diseases are common. Health threats include extreme cold weather, altitude sickness, landslides, and limited access to potable water. Deforestation and the resulting soil erosion and silt load in waterways are rapidly growing environmental problems.

D. India.

1. Geography. India, the seventh largest country in the world and slightly more than one-third the size of the US, has a total land area of 2,973,190 sq km. There are over 7,000 km of coastline on the Arabian Sea, Bay of Bengal, and Indian Ocean. India is bordered on the northwest by Pakistan, on the north by China, Nepal and Bhutan, and on the east by Burma and Bangladesh. The country can be divided into 4 geographic areas: (1) The Himalayas, in northern India, consist of 3 parallel mountain ranges interspersed with large plateaus and valleys. The rugged, steep, perennially snow-covered mountains average 6,700 m above sea level, with a few peaks over 7,620 m. The highest point in India is Kanchenjunga at 8,598 m. Rivers and streams from the mountains flow south to the great plain that contains much of the country's population. (2) The world's largest alluvial plain, the Indo-Gangetic Plain, formed by the Ganges, Indus, and Brahmaputra River Basin systems, is 400 km long and 240 to 320 km wide. (3) The desert region of northwest India, essentially devoid of surface water, contains sand dunes and ridges that average 3 to 8 m above sea level, although some reach 60 m above sea level. (4) The Indian (Deccan) Peninsula, composed of an interior plateau that is mostly 150 to 305 m above sea level, is bordered by the Eastern Ghat Mountains, which average 610 m above sea level, and the Western Ghat Mountains, which vary from 910 to 1,220 m above sea level. About 56% of India's land is arable, and 23% is in forest or woodlands. Overgrazing, deforestation, soil erosion, industrial effluent, vehicle emissions, and desertification are all

growing environmental problems. Tourism is increasing, attracted by India's long and diverse cultural and religious history, associated temples and artifacts, and its relatively large number of native endangered or threatened species. India has 3,319,644 km of highways, of which 1,517,077 km (46%) are paved. The country has many natural resources, including forest products, coal, petroleum, and large deposits of several important mineral ores. However, shortages of funds and technical expertise, and indecisive government policy and management have slowed development of these resources. With a population of over 1 billion people, India has a huge internal market for its own products. Traditional village agriculture is a main part of the economy, employing 67% of the population. Textiles, electric power, machinery, and chemicals are also important products in both internal and export trade. Lack of energy development and poor telecommunications and transportation have delayed improvement of the country's infrastructure and growth of its economy.

2. Climate. India's tropical climate has 3 seasons, with pronounced regional variations. (1) In most of the country, the hot season (April through June) is mostly sunny and dry, with a mean daily maximum temperature of 38 ^{o}C; daytime highs may rise to 49 ^{o}C. Considerably cooler temperatures prevail in the Himalayas. (2) The cool season (October through February) is usually clear and dry in most of India. On the Indo-Gangetic Plain, the mean daily temperature is 7 ^{o}C, with daytime lows occasionally dropping to -2 ^{o}C. In the Himalayas, temperatures fall significantly below freezing. Southern India, with a mean daily minimum of 16 ^{o}C, has a moderate cool season. (3) From June through September, monsoon winds from the southeast and southwest bring almost all of India's annual rainfall. The northeast is typically the wettest part of the country. It and the ocean side of the Western Ghats generally receive more than 2,540 mm of precipitation annually. Cyclones, which form in the Arabian Sea and Bay of Bengal, may cause extensive loss of life, property, crops, and livestock along coastal areas. Mean annual precipitation varies from less than 50 mm in the northwest desert region to 11,400 mm along the Himalayas' northeastern slopes. India's western coastal region receives 1,900 to 3,050 mm of rainfall annually; the east coast receives 840 to 1,270 mm annually. Average annual rainfall on the Indo-Gangetic Plain decreases from 1,190 mm in the south to 250 mm in the north. The Indian Peninsula receives 1,010 to 1,770 mm of rainfall annually. The people of this diverse and densely populated country face serious health risks from severe droughts, flash floods (in major river basins and deltas), severe storms (in mountainous areas and along the coast during monsoon seasons), and earthquakes. Extremes in rainfall and temperature produce great variation in vegetation and species of medically important arthropods.

Delhi (elevation 218 m)

Mean Daily Temperatures (^{o}C)

MONTH	**J**	**F**	**M**	**A**	**M**	**J**	**J**	**A**	**S**	**O**	**N**	**D**
MAXIMUM	22	24	29	36	41	39	35	34	34	34	28	23
MINIMUM	6	9	14	19	26	28	27	26	24	18	11	7
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	25	23	18	5	7	66	211	172	150	30	3	5

Hyderabad (elevation 541 m)

Mean Daily Temperatures (^oC)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	29	32	36	48	40	35	31	31	31	31	29	28
MINIMUM	16	18	21	24	27	24	23	23	22	21	17	15

Monthly Precipitation (liquid equivalent)

MEAN (mm)	8	10	13	31	28	112	152	135	165	64	28	8

Bombay (elevation 11 m)

Mean Daily Temperatures (^oC)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	28	28	30	32	33	31	29	29	29	31	31	29
MINIMUM	20	21	25	25	27	27	26	25	25	25	23	21

Monthly Precipitation (liquid equivalent)

MEAN (mm)	5	5	5	3	15	521	709	440	293	89	20	3

3. Population and Culture. India is a federal republic. It is the second most populous country in the world, with approximately 74% of the population living in rural areas. The annual income for 35% of the population is below the poverty line. The overall population density averages nearly 337 persons per sq km. Roughly 40% of the population is concentrated in the Ganges River Basin, which includes the city of Calcutta. The population's ethnic make-up is roughly 72% Indo-Aryan, 25% Dravidian, and 3% Mongoloids and others. Religious preferences are 80% Hindu, 14% Muslim, 2.4% Christian, 2% Sikh, 0.7% Buddhist, 0.5% Jains, and 0.4% other. Every year the Maha Kumbh Mela, a 41-day religious festival, draws millions of Hindus to the delta where the Ganges and Yamuna Rivers meet near the city of Allahabad. In the year 2001 an estimated 70 million pilgrams will will engage in a mass ritual bath in the Ganges. The rite is known as a snan and is considered the spiritual experience of a lifetime. Although the Ganges is badly polluted, people freely drink and wash in the water. It is the perfect opportunity for the outbreak of many infectious diseases. English is the most important language for national and commercial communication. Hindi is the national language and primary tongue of 30% of India's people. At least 24 different languages, each spoken by a million or more persons, and numerous dialects are spoken throughout India. The Hindu caste system and strong religious and family ties have led to frequent local and regional fighting. Ethnic-based fighting has occurred throughout Indian history but has increased in frequency and scope during the past 2 decades. Total population one billion; 34% urbanized; literacy rate 52%.

4. Sanitation and Living Conditions. Water sources include surface and groundwater. Although India is one of the wettest countries in the world, 75% of the annual precipitation falls during the monsoon season, and it is not efficiently collected and stored. In addition, there is great regional variation in water supplies. Water is seasonally scarce on the plateau of the Indian Peninsula and permanently scarce in the desert. Significant water shortages occur in many areas, and disputes over water resources occur on India's borders. Groundwater is becoming saline in coastal areas because of salt water intrusion, and aquifers in the northwest states of Punjab, Delhi, Haryana, Rajasthan, and Gujarat are being rapidly depleted. The few water treatment systems that exist in India are characterized by poor maintenance, intermittent power

supplies, inadequate treatment, and back siphonage that result in contaminated water in many areas. In urban areas, standpipes are the most common method of distribution. Rural areas collect water directly from community wells or raw surface sources. India has some of the poorest sanitary conditions in the world. Municipal sewage collection systems are vastly inadequate for the large population. Only 8 of India's 3,119 cities and towns have adequate sewage treatment systems; another 209 have partial treatment facilities. Only 10% of the sewage receives treatment in the main cities. Most sewage flows untreated, directly into rivers, lakes or other water sources. Indiscriminate human defecation is common, and open drains carry human and animal wastes into rivers and streams. There is no solid waste collection except in affluent urban areas. Open dump sites are common in populated areas. Rural sanitation is almost nonexistent. These conditions have led to increased rodent populations and the threat of rodent-borne disease. Hordes of flies breeding in city garbage and excrement help spread gastrointestinal disease. Night soil is a commonly used fertilizer. Poor food sanitation is widespread, even in large cities and in many modern restaurants. Food-borne and water-borne diseases and parasites are common. Amebiasis infection rates up to 84% have been reported in northern India. Giardiasis infection rates of 21% to 62% have been reported from many areas.

India's annual urban growth rate exceeds 3.5%. The urban population increased by nearly 150% from 1975 to 2000 with over one third of India's people crowded into cities and shantytowns. By the year 2000, India is expected to have at least 40 cities with populations each exceeding 1 million. In 1972 the population of Bombay was 6 million. By the mid 1980s, the city was attracting 350,000 migrants each year. By the year 2000, there will be an estimated 18 million people living in Bombay, with over 1 million people sleeping in the streets. Urbanization has been accompanied by increased poverty, overcrowding, malnutrition, decreased sanitation, and industrial stress. These conditions promote the transmission of many infectious diseases and some vector-borne diseases, such as plague, leptospirosis, and epidemic and murine typhus. Malnutrition and physical stress increase the population's susceptibility to disease as well as the severity of symptoms. The potential for dengue outbreaks has increased in urban areas where susceptible populations living at high density are creating breeding sites for *Aedes aegypti*. Populations of *Culex pipiens* have greatly increased along with urbanization, leading to increased risk of filariasis and transmission of mosquito-borne viruses. Increased breeding sites for *Anopheles stephensi* have resulted in urban transmission of malaria, a primarily rural disease. When urban areas sprawl into agricultural land, forests and fields, the satellite communities and shantytowns frequently intrude into zoonotic cycles of disease. Efforts to improve the quality of life in urban slums have met with little success. The Indian government is trying to redirect urban growth away from such huge cities as Calcutta, Delhi and Bombay, to smaller towns where the problems of urbanization can be better managed.

Urbanization is usually accompanied by industrialization. Industries often become established on the outskirts of towns and attract laborers and craftsmen from rural areas. These immigrants may become infected with arthropod-borne disease when they visit relatives in their home villages. In this way, diseases often spread from rural to urban areas. Increases in transmission of filariasis have clearly been linked to urbanization. In addition, industrial pollution and chemical contamination of water, food, air and soil are serious environmental problems in India. High levels of arsenic (up to 3.7 mg/L) associated with skin blistering and fluoride levels up to 11.5 mg/L in groundwater have been found in the West Bengal and Kanpur areas, respectively. Industries in the Manzira River basin near Hyderabad have surface and groundwater contaminated with arsenic, copper, selenium and boron over a wide area. Dissolved salts in groundwater occur in the state of Uttar Pradesh. Emissions from vehicles, industries, and thermal power plants that burn a high-ash coal expose residents of most cities to sulfur dioxide, nitrogen oxides, lead, fly ash, and suspended particulates. The levels of particulates reportedly have exceeded the WHO standard in as many as 14 cities; in Calcutta, particulate standards are exceeded nearly two-thirds of the year. Extensive air pollution results from forest fires and the burning of cow dung and agricultural wastes for fuel.

E. Maldives.

1. Geography. The Maldives is an archipelago of 1,190 coral islands grouped into 26 atolls, in the Indian Ocean southwest of India. The islands are roughly centered on the 73rd degree East longitude line and stretch north to south, from about one degree South latitude to about eight degrees North latitude. Most of

the atolls are surrounded by outer reefs, or *faros*. Their total land area is about 300 sq km, roughly 1.7 times the size of Washington, DC. The average height of these islands is only about 1.3 m elevation, and about 80% of the land is less than one m above sea level. The highest point of land is an unnamed point 2.4 m above mean sea level on Wilingili Island in the Addu Atoll, approximately one degree of latitude below the equator. Only 200 islands are inhabited. About 10% of the land is arable, 3% is pasture, and 3% is forest or woodlands. These islands are very vulnerable to flooding from rises in sea level and from storms. A sea wall has been built around the capital island of Malé, in the Malé Atoll. Tourism, fishing, garment making, handicrafts, boat building, and coconut production are the main industries. The British maintained a small military presence at a staging point on Gan in the southernmost atoll, Addu, during most of the period from World War II until 1976. Current and future sources and reserves of fresh drinking water are a great concern.

2. Climate. The Maldives have a tropical climate with abundant rainfall (annual average of 1613 mm, mainly falling during June through August, primarily in the northern islands) and high temperatures throughout the year. The northern islands are occasionally affected by violent storms caused by tropical cyclones. Most rain falls in the southern islands, from November to March each year.

Minnicoy (elevation 3 m)

Mean Daily Temperatures (^oC)

MONTH	**J**	**F**	**M**	**A**	**M**	**J**	**J**	**A**	**S**	**O**	**N**	**D**
MAXIMUM	29	29	30	31	31	30	29	29	29	29	29	29
MINIMUM	23	24	25	27	26	25	24	25	25	24	23	23
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	46	18	23	58	178	295	226	198	160	185	140	86

3. Population and Culture. The native population of the Maldives is slightly more than 330,000 (a density of 1,100 persons per sq km). About 25% of the native people live on the capital island of Malé. Because of historically severe shortages of skilled labor, there are estimated to be more than 12,000 guest workers, mainly from Sri Lanka and India, currently living in the Maldives. This is a strictly Muslim nation, with nearly 100% of natives belonging to the Sunni sect, and nearly all are Dhivehi (Maldivian) in ethnic origin, with a few Arabs, Sinhalese, Dravidians, and Africans included. Wealth, power, political involvement, and access to health care and modern facilities and goods are limited to an elite few and their families, nearly all of whom live on Malé. Most staple goods, including food, petroleum, and manufactured items, must be imported into the Maldives. People on most of the other islands are generally poor and lack most goods and services. The standard of living for the inhabitants of Addu Atoll declined significantly after the British Royal Air Force closed an air staging station on the island of Gan in 1976. Development of tourist resorts on a few islands has had little economic impact on the bulk of the native population. Nonetheless, the population is 93% literate and about 26% urbanized.

4. Sanitation and Living Conditions. Some water is caught during rains, but most drinking water is taken from freshwater aquifers, and these are being rapidly depleted. There are no desalinization plants currently in use or even planned for the near future. There are no reliable potable water distribution systems on most islands, and the vast majority of the population does not have access to sewage treatment. Risk of food-borne and water-borne disease is high. Only on the capital island are water and sewage treatment facilities commonly available, and access is largely limited to wealthy residents or foreign tourists. Waste disposal is often indiscriminate and trash is dumped or burned in the open on most outlying islands. Abundant rodent, fly and mosquito populations thrive under these conditions. The majority of the people live in crudely built huts or flimsy shelters on most islands except Malé. Lack of medical facilities makes the detection, diagnosis, and treatment of disease outbreaks difficult.

F. Nepal.

1. Geography. Nepal is among the poorest and least developed countries in the world. It is landlocked and is bordered by China and India. With a total land area of 136,800 sq km, it is about the size of Arkansas. The country is not wider than 225 km at any point. Despite its small size, Nepal exhibits great geographic diversity and can be divided into 3 regions: (1) The Himalayan mountain range in the north, covering one-third of Nepal, has elevations between 4,900 and 8,800 m above sea level. The highest point in the country is Mt. Everest, at 8,848 m; the lowest point is at Kanchan Kalan at 70 m. Rivers flowing from the High Himalayas to the Ganges Plain have carved out valleys with strips of relatively flat land that can be farmed. (2) The Tarai region, which borders India and is part of the fertile Ganges Plain, is a narrow belt of flat mashy land about 40 to 180 m above sea level. It is famed for its jungles and wildlife. The slopes north of the Tarai, known as the Hills, are inhabited to an elevation of about 2,500 m and used in summer for grazing to about 4,000 m. (3) The Kathmandu Valley is an extensively cultivated fertile basin north of the Tarai, with 560 sq km of total land area lying at an elevation over 1300 m. The land is 17% arable, 15% permanent pasture, and 42% forests and woodland. Agriculture is the most important industry, employing about 80% of the population. Textiles and carpets make up most of Nepal's current exports. Tourism is increasing as a major source of foreign revenue. Nepal contains 8 of the world's 10 highest mountain peaks, including the highest, Mt. Everest. There is much potential to develop hydroelectric power, but limited resources and internal political instability have slowed progress. Firewood is the primary source of fuel. Cutting trees without replanting, together with agricultural expansion, have caused widespread deforestation and soil erosion.

2. Climate. Climatic conditions in Nepal vary by geographic region. (1) In the northern mountainous region, winters (September through May) are long and cold; temperatures rarely rise above 0 °C. The short summers (June through August) have a mean daily temperature of 20 °C, although permanent frost prevails at elevations above 5,000 m. Summer brings most of the region's 500 mm of annual precipitation. (2) The Tarai region has a hot, humid rainy season (June through September), a cool season (October through February), and a dry, warm season (March through May). Seasonal temperatures are similar to those in the Kathmandu Valley. Annual rainfall in the Tarai averages 700 mm. However, some areas of eastern Tarai frequently receive as much as 5,000 mm per year. (3) The Kathmandu Valley has a subtropical climate with three seasons. The hot season lasts from May through June, with a mean daily temperature of 30 °C. The monsoon season (July through September) brings violent thunderstorms and periodic flooding. Most areas of the Kathmandu Valley receive 1,520 mm of annual rainfall. The cold season (October through April) has a mean daily minimum temperature of 2 °C. Daily temperature fluctuations of 11 to17 °C occur in most of Nepal's interior regions. Storms can develop quickly, especially in the mountains, and are often severe enough to cause local flash flooding and landslides.

Kathmandu (elevation 1,338 m)

Mean Daily Temperatures (°C)

MONTH	**J**	**F**	**M**	**A**	**M**	**J**	**J**	**A**	**S**	**O**	**N**	**D**
MAXIMUM	18	19	25	28	30	29	28	28	28	26	23	19
MINIMUM	1	3	7	11	16	19	20	20	18	13	7	2
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	15	41	23	58	122	246	373	345	155	38	8	3

3. Population and Culture. Nepal is one of the least urbanized countries in Asia. It is the only officially Hindu country in the world. The population is unevenly distributed. While the barren highlands are virtually uninhabited, the fertile Kathmandu Valley exceeds 356 residents per sq km. Overall population density is 177 persons per sq km. It was estimated in 1999 that nearly half of the people live below the poverty line. The population is made up of many different ethnic groups, including Newars, Indians,

Tibetans, Gurungs, Magars, Tamangs, Bhotias, Rais, Limbus, and Sherpas. Sharp ethnic distinctions have been lost due to years of intermingling. The population's religious preferences are 90% Hindu, 5% Buddhist, 3% Muslim, and 2% other, although there is no clear distinction between many Hindu and Buddhist groups. An issue of major concern is the presence of about 91,000 refugees from Bhutan, 90% of whom are now living in 7 camps that are being run by the U.N. Office of the High Commissioner for Refugees (UNHCR). Total population 24 million people; 12% urbanized; literacy rate 27.5%.

4. Sanitation and Living Conditions. Nepal's water sources are primarily in the Himalayas. Demand for groundwater exceeds supply, resulting in water table reductions of as much as 2.5 m per year. Groundwater in many areas is contaminated with sewage. Urban water treatment for most of Nepal is limited to filtration and chlorination. Intermittent and chronic water shortages result in low pressure, which reduces water distribution and subjects water to infiltration from sewage. Within Kathmandu, about 80% of the population has access to treated water that includes public standpipes and individual household hookups. Most rural inhabitants obtain water directly from rivers and springs, or neighborhood wells. Basic sanitation services are lacking throughout the country, and untreated human and animal waste is a serious public health threat in Nepal. Only Kathmandu has a sewage system, and it serves only about 25% of the city's inhabitants. The Bagmati River in Kathmandu Valley is heavily polluted with raw sewage. Typhoid fever is highly endemic, and hepatitis E is widespread, accounting for 85% of cases of acute viral hepatitis. The risk of cholera is considered low. Other sources of water contamination include wastes from various industries, including leather, textiles, paper, batteries, soap, and distilleries. Chemical fertilizer and pesticide residues in agricultural runoff also contaminate surface waters. In Kathmandu, suspended particulate emissions in the air have become a growing problem because of the increasing number of vehicles and the unregulated growth of industries, such as cement factories and brick kilns. There is little or no urban or rural refuse collection in Nepal; most refuse is disposed of indiscriminately. Kathmandu's solid waste landfill is near capacity, and solid wastes are dumped directly into the Vishumati River. Over 300,000 tourists visit Nepal each year, which contributes to a growing refuse and sewage disposal problem.

G. Pakistan.

1. Geography. Pakistan, with approximately 778,700 sq km of total land area, is about twice the size of California. It is bordered on the northwest by Iran and Afghanistan, on the northeast by China, on the southeast by India, and on the southwest by a 1,046 km coastline on the Arabian Sea. It controls both the Khyber Pass and the Bolan Pass, two traditional invasion routes between Central Asia and the Indian Subcontinent. It has four major geographic regions: (1) The northern one-third of Pakistan consists of the Himalayan and Trans-Himalayan mountain ranges, with a mean elevation of approximately 6,100 m, and several peaks exceeding 8,000 m. The highest point in the country is Mt. Godwin Austen (K2) at 8,611 m. (2) The Balochistan Plateau in western Pakistan, a broad, arid tableland between 305 and 914 m above sea level, is interspersed with northeast to southwest mountain ridges. This thinly populated area has some irrigation and farming, but most inhabitants are Baluchi nomads who call the region Makran. (3) Geographically, the 518,000-sq km Indus Plain in south and central Pakistan is the country's most prosperous agricultural region and is the heart of Pakistan. Within the vast Indus Plain stretching from the Punjab to the coast near Karachi, the Indus River and its 5 lower tributaries form southern Pakistan's fertile alluvial basin. Irrigation from the rivers and the system of connecting canals supports intensive farming. (4) The Thal, Cholistan, and Thar Deserts, all extensions of the Thar Desert of western India, cover southeast Pakistan. Natural resources include extensive natural gas reserves, some petroleum, and several mineral ores, but lack of funding, poor government planning and political instability have hampered the development of these resources. Land uses include 27% arable, 6% pasture, and 5% forests and woodlands. Cotton, rice and textiles are important exports. Pakistan has limited fresh water resources, and deforestation, soil erosion and desertification are growing environmental problems.

2. Climate. Pakistan has a climate that is mostly hot, dry desert with temperate areas in the northwest and arctic conditions in the north. It has significant diurnal, regional, and seasonal temperature variations. Daily variations of 11 ^{o}C to 17 ^{o}C occur in most of Pakistan's interior regions. During the dry, cool winter (December through February), mean daily temperatures of 14 ^{o}C occur on the Indus Plain, 20 ^{o}C along the coast, and -20 ^{o}C in the northern mountains. During the summer (March through May), mean daily temperatures vary from 29 ^{o}C along the coast to 35 ^{o}C in the desert region and on the Indus Plain, and 0 ^{o}C

in the mountains. The monsoon seasons, southwesterly during June through July and northeasterly during October through November, bring periodic flooding and deliver an annual average rainfall of 1,520 mm to the northern highlands, 127 mm in the Balochistan Plateau, 380 mm in the river valleys, 150 to 200 mm along the coast, and 100 mm in the desert region. Humans can face serious health risks from extremes of heat and cold (as high as 53 °C in Jacobabad; as low as -20 °C in the northern mountains), altitude sickness, dust and sand storms, occasionally severe earthquakes, and flooding along the Indus River after heavy rains in July and August.

Karachi (elevation 4 m)

Mean Daily Temperatures (°C)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	25	26	29	32	33	33	32	31	31	32	30	26
MINIMUM	12	14	19	22	26	27	27	26	25	22	17	13
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	13	10	8	3	3	18	81	41	13	0	3	5

3. Population and Culture. Population density varies from 1 person per sq km on the Balochistan Plateau to more than 200 persons per sq km in parts of Sind and Punjab Provinces (averaging about 177 persons per sq km countrywide). The high rural-to-urban migration rate (approximately 5% annually) compounds deteriorating living conditions in most cities. Karachi has about a 6% annual growth rate. The ethnic make-up of the population is about 50% Punjabi, 15% Pashtun (Pathan), 15% Sindhi, 8% Mohajir (immigrants from India and their descendants), 5% Baloch, and 7% other. Religious preferences are: Muslim 97% (Sunni 77%, Shi'a 20%), 3% Christian, Hindu, and others. About 47% of the people are involved in agriculture. Cultural and political ties along the lines of the extended family, clan, caste, and religious group are often very strong. There is much internal strife and violence between different ethnic groups, religious sects, and nationalities (especially between foreign refugees and native Pakistanis). Islamic laws and customs have led to serious repression of womens' rights and restrictions of public freedoms. There are great extremes of wealth between the elite few rich people and the masses of poor. Availability of medical doctors, hospital beds, medical equipment, and medicines in Pakistan is among the lowest in the world. Total population 138 million; 33% urbanized; literacy rate 38%.

4. Sanitation and Living Conditions. Perennial and intermittent surface waters, ground water and irrigation canals are water sources. Water shortages occur seasonally on the Indus Plain and continuously over most of the Balochistan Plateau and in desert regions. Pakistan's rapidly growing urban population is placing severe demands on limited water sources. Less than 50% of all urban dwellers and only 5% of rural inhabitants have access to municipal water. In Karachi, piped water is available for only a few hours a day. While many large cities maintain minimal water treatment, the methods used are inadequate. Aging distribution systems are poorly maintained, and power outages subject pumping operations to low pressure and possible sewage infiltration. While wealthier sections of cities may have individual house hookups, most urban inhabitants collect water from standpipes. Rural dwellers obtain water directly from untreated sources. The standard of living is far below that of the West. In most modern sections of large cities, sewage systems serve less than 5% of the population. Septic tanks and pit and bucket latrines are used in some urban areas, but indiscriminate waste disposal is common. Most sewers are connected to open waterways that run into main rivers. In large cities, refuse is collected sporadically and dumped in open pits outside the city. Improper food sanitation occurs countrywide. Food-borne and water-borne diseases are common throughout the area, including cholera (with the vaccine-resistant Bengal strain being reported), the dysenteries, typhoid fever, viral hepatitis, and parasitic helminthic intestinal infections. There may be some health risks of local significance from industrial or chemical contamination of water, food, air, or soil from such industries as tanning, textiles, electroplating, brick kilns, pesticide production, steel mills, diesel generators, and cement factories. Vehicles and industrial operations often discharge large

quantities of particulate matter into the air of urban areas. Biomass (cow dung, wood, leaves) burning also contributes to local air pollution.

H. Sri Lanka.

1. Geography. Sri Lanka includes one large pear-shaped island and several smaller coral islets (Adam's Bridge) in the Indian Ocean, just southeast of the southern tip of India. It contains approximately 64,740 sq km of land area, 1,340 km of shoreline, and is about the size of West Virginia. It can be divided into two geographic regions: (1) A gently rolling plain covers the northern half of the island and parts of the southern coastal region. (2) The south-central part of Sri Lanka is hilly and mountainous, with elevations between 900 and 2,500 m above sea level. The highest point is Pidurutalagala Mountain at 2,524 m above sea level. Sixteen large rivers radiate from the center of the island and provide a good drainage network; the largest and longest rivers are the Mahaweli Ganga (330 km) and the Aruvi Aru (170 km). Land use includes: 14% arable, 15% crops, 7% permanent pasture, and 32% forested. Water source depletion and soil erosion are problems in some locations. Although Sri Lanka is still the world's largest exporter of tea, textiles and garments now make up 63% of the country's exports, while tea has dropped to only 20%.

2. Climate. Sri Lanka has a tropical climate with seasonal monsoons. Temperatures countrywide are similar to those recorded at Colombo, and humidity is typically 70% or greater. High temperatures may reach 40 ^{o}C. From June through September, the monsoons arrive from the southwest. The southern highlands force the humid summer monsoon winds to rise, producing heavy rainfall in the southwestern quarter of the island. From December through March, the monsoons arrive from the northeast, causing countrywide but less predictable precipitation. Rain can be quite sudden and heavy, causing damaging floods. Annual rainfall varies regionally. The southwestern plain and uplands receive approximately 2,540 to 5,080 mm, while the north, central, and southeastern plains receive approximately 1,270 to 1,900 mm. Cyclones and tornadoes occur infrequently but have caused much loss of property and lives.

Colombo (elevation 5 m)

Mean Daily Temperatures (^{o}C)

MONTH	J	F	M	A	M	J	J	A	S	O	N	D
MAXIMUM	30	31	31	31	31	30	30	30	30	30	30	30
MINIMUM	23	24	25	26	27	26	26	26	26	25	24	24
Monthly Precipitation (liquid equivalent)												
MEAN (mm)	89	69	47	231	371	224	35	109	160	48	15	147

3. Population and Culture. The population of Sri Lanka is predominately rural and poor. Population density is greatest in the southwest quarter of the island, with approximately 22% of the total population living in the Colombo District. Nearly 44% of the population of Colombo lives in slums or squatter settlements. Overall average population density is 295 persons per sq km. Sri Lanka has one of the lowest per capita incomes in the world, with 35% of the population living below the poverty line. Ethnic make-up of the population is about 74% Sinhalese, 18% Tamil, 7% Moor, and 1% a mixture of Brugher, Malay, and Vedda. Fighting between the Sinhalese majority and Tamil minority has taken 50,000 lives over the past 15 years and threatens political stability. The Sinhalese majority are concentrated in the southern half of the island, while the Tamils live almost exclusively in the northern portion, principally on or near the Jaffna Peninsula. Religious preferences of the population are 69% Buddhist, 15% Hindu, 8% Christian, and 8% Muslim. Typically, cultural, family, ethnic and religious ties are very strong and greatly influence people's daily lives. Total population 19 million; 22% urbanized; literacy rate 90%.

4. Sanitation and Living Conditions. Surface water sources include rivers and a major network of irrigation canals. Tube wells and dug wells provide additional water sources. Water piped through municipal treatment and distribution systems is available only in large urban areas. Treatment plants are

inadequate, and contamination of the distribution systems occurs because of leaks, back siphonage, and cross-contamination. Rural residents obtain water directly from untreated sources. Sanitary conditions, including food sanitation, are below Western standards. Public sanitation services are inadequate throughout Sri Lanka. Although 73% of urban and 56% of rural residents have access to in-house or in-compound septic tank or pit latrines, indiscriminate defecation is common, and open drains carry untreated human and animal wastes into rivers and streams. Only Colombo has a sewer system, and it collects and disposes raw sewage directly into the ocean. Refuse is not collected regularly or sent to landfills. Open dumps are common in Colombo and other cities. Night soil is commonly used as a fertilizer. Consequently, food-borne and water-borne diseases are common throughout the island. Contamination of water, air and soil from agricultural chemicals and pollution from industries such as paper mills, cement factories, and brick and tile works are growing environmental problems.

V. Militarily Important Vector-borne Diseases with Short Incubation Periods   (<15 days)

A. Malaria.

Human malaria is caused by any of 4 protozoan species in the genus *Plasmodium* that are transmitted by the bite of an infective female *Anopheles* mosquito. Clinical symptoms of malaria vary with the species. The most serious malaria infection, *falciparum* malaria, can produce life-threatening complications, including renal and hepatic failure, cerebral involvement and coma. Case fatality rates among children and nonimmune adults exceed 10% when not treated. The other human malarias, *vivax*, *malariae* and *ovale*, are not life-threatening except in the very young, the very old, or persons in poor health. Illness is characterized by malaise, fever, shaking chills, headache and nausea. The periodicity of the fever, occurring daily, every other day, or every third day, is characteristic of the *Plasmodium* species. Nonfatal cases of malaria are extremely debilitating. Relapses of improperly treated malaria can occur years after the initial infection in all but *falciparum* malaria. *Plasmodium malariae* infections may persist for as long as 50 years, with recurrent febrile episodes. Persons who are partially immune or have been taking prophylactic drugs may show an atypical clinical picture. Treatment of malaria has been complicated by the spread of multiple drug-resistant strains of *P. falciparum* in many parts of the world.   Current information on foci of drug resistance is published annually by the World Health Organization (WHO) and can also be obtained from the Malaria Section of the Centers for Disease Control and Prevention, and the Armed Forces Medical Intelligence Center.

Military Impact and Historical Perspective. Malaria has had an epic impact on civilizations and military operations. During the U.S. Civil War, one-half of the white troops and four-fifths of the black troops in the Union armies contracted malaria annually. During World War I, in the Macedonian campaign, the French army was crippled with 96,000 cases of malaria. In 1918, over 2 million man-days were lost in the British Macedonian Army because of malaria. During World War II, malaria caused five times as many US casualties in the South Pacific as did enemy action. The highest annual incidence rate of malaria during World War II (98.5 cases per 1,000) occurred in the China-Burma-India theater. In 1942, malaria was the major cause of casualties in General Stilwell's forces in North Burma. There were 9,160 cases of malaria in the China-Burma-India theater during 1943, with nearly 115,000 man-days lost. At one time, 55% of the beds in the 20th General Hospital, located near Ledo in Assam, were occupied by patients with malaria. An epidemic of malaria occurred during 1944 at the 1306th Air Force Base Unit, Air Transport Command, located in Karachi, India (Pakistan). The incidence rose rapidly to 1,202 cases per 1,000 per annum during the first two weeks of October, but dropped to 148 per 1,000 during the first two weeks of November following the institution of malaria control measures. There were approximately 81,000 confirmed cases of malaria in the US Army in the Mediterranean Theater from1942 to1945. The average length of hospitalization for malaria in 1943 was 17 days, representing a total of 425,000 man-days lost during the year, or the equivalent of an entire division lost for a month. In 1952, during the Korean War, the 1st Marine Division suffered up to 40 cases per 1,000 marines. Battle casualties accounted for only 17% of American hospitalizations during the Vietnam War. Many regiments were rendered ineffective due to the incidence of malaria, and many US military units experienced up to 100 cases of malaria per 1,000 personnel per year. Elements of the 73rd Airborne Brigade had an incidence of 400 cases of malaria per 1,000 during 1967 and early 1968. Almost 300 military personnel contracted malaria during Operation Restore Hope in Somalia. Malaria remains a threat to military forces due to widespread drug resistance in plasmodia, insecticide resistance in the vectors, and the consequent resurgence of malaria in many areas of the world.

Disease Distribution. Endemic malaria has been eradicated from most temperate countries, but it still is a major health problem in many tropical and subtropical areas. Worldwide, there are an estimated 250 to 300 million cases of malaria annually, with 2 to 3 million deaths. The WHO estimates that in Africa nearly 1 million children under the age of 10 die from malaria every year. Globally, *P. falciparum* and *P. vivax* cause the vast majority of cases. *Plasmodium falciparum* occurs in most endemic areas of the world and is the predominant species in Africa. *Plasmodium vivax* is also common in most endemic areas except Africa. *Plasmodium ovale* occurs mainly in Africa, and *P. malariae* occurs at low levels in many parts of the world. In most endemic areas the greatest malaria risk is in rural locations, with little or no risk in

cities. However, in Somalia during Operation Restore Hope (1993), several malaria cases occurred in troops who were only in Mogadishu. In India the transmission of malaria in urban areas is a serious public health problem. The distribution of malaria is depicted in Figure 1.

Figure 1. Distribution of Malaria in South Central Asia.

FIG. 1. DISTRIBUTION OF MALARIA IN SOUTH CENTRAL ASIA (DARK SHADING).

Afghanistan: Malaria is endemic at high levels countrywide. Malaria cases are almost exclusively caused by *P. vivax*; however, *P. falciparum* infections are increasing and currently account for up to 2% of cases. Most cases of falciparum malaria have occurred in eastern Afghanistan along the border with Pakistan. There is no risk of malaria in Kabul, but malaria may occur in urban areas of southern Afghanistan. Transmission is limited to warmer months of the year from May through October below 2,000 m elevation, with peak transmission occurring during August and September. Malaria control and surveillance activities have been limited due to ongoing civil conflict.
Chloroquine-resistant falciparum malaria has been reported from areas bordering Pakistan.

Bangladesh: Malaria transmission occurs year-round throughout the country except for the city of Dhaka. Risk of transmission is elevated during the monsoon season (usually July through September) and in the forested areas and foothills of the northeastern border areas with India and the southeastern border areas with Burma. *Plasmodium falciparum* accounts for 50% to 70% of malaria cases and *P. vivax* accounts for the remainder of cases. Chloroquine resistance has become widespread in Bangladesh since the first report of resistance in the country in 1971. Resistance of *P. falciparum* to sulfadoxine-pyrimethamine (Fansidar™) has also been reported, and resistance to quinine and mefloquine may also occur.

Bhutan: Risk of malaria is present year-round in the south and southeastern districts bordering India. Risk is generally limited to the southern Duars Plain and mountain valleys of the Lesser Himalayas below 1,700 m elevation in the Chirang, Geylephug, Samchi, Samdrup Jongkhar, and Shemgang Districts. Risk is elevated during the rainy season from May through October. *Plasmodium falciparum* accounts for 45% of the cases, with *P. vivax* accounting for the remainder. Chloroquine and Fansidar™-resistant strains of *P. falciparum* have been reported.

India: In 1947 India had 75 million malaria cases and 750,000 deaths. The Indian National Malaria Control program was initiated in 1953 after local success with residual DDT spraying from 1948 to 1952. By 1965 malaria cases had fallen to fewer than 100,000. The Indian public health system and malaria control programs began to deteriorate by the early 1970s just as drug-resistant malaria strains and insecticide -resistant vectors were becoming widespread. Reported malaria cases in 1976 were more than 6.5 million, but were possibly as high as 25 million. Malaria is no longer a reportable disease and surveillance is passive. Essentially the only malaria slides taken are those from patients who report to clinics with fever. In 1995 there were an estimated 15 million cases with as many as 20,000 fatalities. More than 25,000 cases of malaria were reported in Calcutta alone. India's current malaria control strategy is based on diagnosis and chemotherapy of active cases. Unfortunately, this has contributed to the spread of multidrug-resistant strains of *Plasmodium*.

Currently, risk of malaria is present countrywide below 2,000 m elevation, including urban areas. The states of Himechel Pradesh, Jammu and Kasmir, and Sikkim are considered risk free. *Plasmodium vivax* predominates and accounts for nearly 60% of all cases. *Plasmodium falciparum* accounts for most of the remaining cases, although scattered cases of *P. malariae* and *P. ovale* have been reported. *Plasmodium falciparum* is more common in the eastern and northeastern states. Malaria transmission occurs year-round in the tropical cities of Bombay, Calcutta, and Madras. Malaria in the more temperate city of New Delhi is seasonal, with most cases occurring from May through October. Urban malaria has been increasing in recent years as a result of uncontrolled urban growth, migration of infected workers from rural to urban areas, and inadequate control programs. Urban malaria is largely a manmade problem. Construction sites provide ideal breeding conditions (ditches, puddles, etc.) for *An. stephensi*. Wells, cisterns, water storage tanks throughout urban areas, and many other artificial containers found in slum areas, provide additional breeding sites. The picturesque city of Mangalore in southern India is typical. Malaria was unknown until 1990, when the area experienced a great increase in construction due to urbanization and industrialization. Malaria cases increased from 1 or 2 cases per month in 1990 to nearly 30,000 in 1997.

Chloroquine-resistant falciparum malaria was first reported in 1973 from Assam State in northeastern India. Chloroquine-resistance now exists countrywide, including some highly resistant strains of *P. falciparum*. Several hundred deaths occurred in Rajasthan State in 1994 due to chloroquine-resistant malaria. Fansidar™ resistance has been reported from Orissa State, and resistance to pyrimethamine and amodiaquine has been reported in falciparum malaria cases in northeastern India. More troubling are reports of
resistance to chloroquine and primaquine treatment by *P. vivax* in Bombay and possibly in other areas of India.

Nepal: Malaria primarily occurs in rural areas below 1,200 m elevation in the southern Terai plain districts of Bara, Dhanuka, Kapilvastu, Mahotari, Parsa, Rautahat, Rupandehi, and Sarlahi. Cases of malaria have been reported from urban areas and at elevations up to 2,100 m. Risk of transmission is highest along the Indian border. Kathmandu and the northern Himalayan districts are considered malaria-free. *Plasmodium vivax* accounts for 90% of cases, with *P. falciparum* accounting for the remainder. Transmission occurs

year-round but usually peaks during the monsoon season from July through October. Chloroquine-resistant falciparum malaria has been reported primarily along the Indian border but may occur in other malarious areas of Nepal.

Maldives: The Maldive Islands are considered malaria-free.

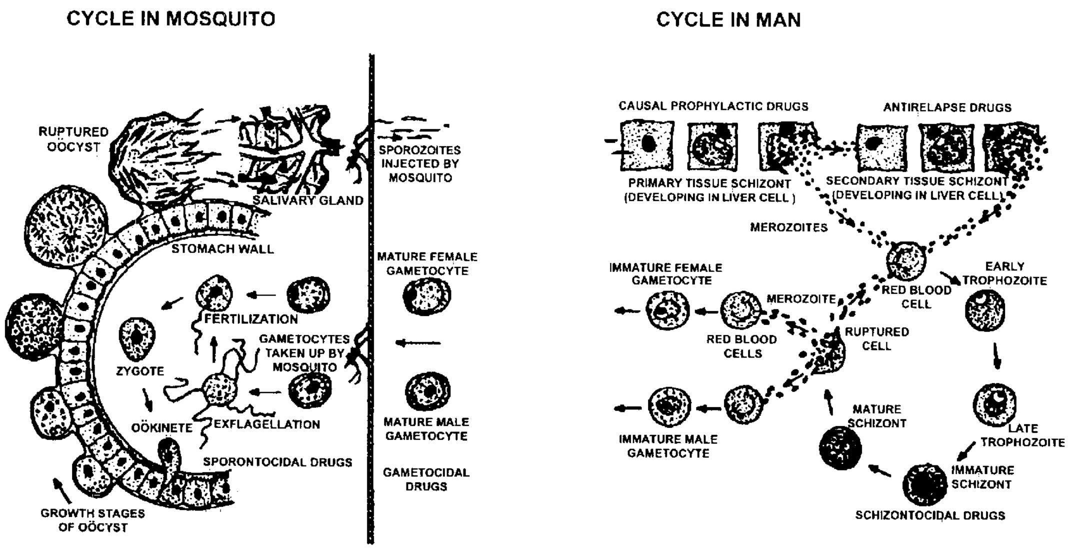
Pakistan: Malaria transmission occurs countrywide in rural areas and at the fringes of most urban areas, including Karachi. Punjab is the most malarious area of the country. Transmission usually occurs at elevations below 2,000 m, but malaria cases have been reported up to 3,500 m elevation in the Gilgit Agency in the north. *Plasmodium falciparum* causes 75% of malaria cases in Baluchistan and Sind Province, 35% of infections in Punjab Province, and 15% of the cases in N.W. Frontier Province. *Plasmodium vivax* accounts for the remainder. Transmission occurs year-round, although vector abundance is highest from May through November. Chloroquine-resistance occurs countrywide, and Fansidar™-resistance has been reported from Punjab. High levels of resistance to both therapeutic agents have been reported in Pakistani and Afghan refugee settlements in western Pakistan.

Sri Lanka: A resurgence of malaria has occurred in this island nation largely as a result of the Maheweli River Basin irrigation project . Only 17 cases were recorded in 1963. By 1987 over 412,000 cases had been recorded. Currently, malaria occurs countrywide, including most urban areas, below 800 m elevation. The districts of Colombo, Kalutara and Nuwara Eliya are free of malaria. The highest incidence of malaria occurs in the area around Anuradhapura and in the northern and southeastern portions of the island. Malaria is less common in the Jaffna Peninsula and southwestern Sri Lanka. Falciparum malaria accounts for up to 30% of cases, with *P. vivax* causing the rest. This is notable because Sri Lanka was known for having virtually no *P. falciparum* infections in the past. Highly chloroquine-resistant strains of falciparum malaria are now widespread.

Transmission Cycle(s). Humans are the only reservoir host of human malaria. Nonhuman primates are naturally infected by many *Plasmodium* species that can infect humans, but natural transmission is rare. Female mosquitoes of the genus *Anopheles* are the exclusive vectors of human malaria. *Plasmodium* species undergo a complicated development in the mosquito. When a female *Anopheles* ingests blood containing the sexual stages (gametocytes) of the parasite, male and female gametes unite to form a motile ookinete that penetrates the mosquito's stomach wall and encysts on the outer surface of the midgut. Thousands of sporozoites are eventually released, and some of these migrate to the salivary glands. Infective sporozoites are subsequently injected into a human host when the mosquito takes a blood meal (Figure 2). The time between ingestion of gametocytes and liberation of sporozoites, ranging from 8 to 35 days, is dependent on the temperature and the species of *Plasmodium*. Malaria parasites develop in the mosquito vector most efficiently when ambient air temperatures are between 25EC and 30EC. Parasite development is prolonged during cool seasons and at high altitudes, and may exceed the life expectancy of the vector. Adult vector life span varies widely depending on species and environmental conditions. Longevity is an important characteristic of a good vector. Once infected, mosquitoes remain infective for life and generally transmit sporozoites at each subsequent feeding. Vector competence is frequently higher with indigenous strains of malaria. This may decrease the likelihood that imported strains from migrants will become established.

Figure 2. Life Cycle of *Plasmodium*, the Malaria Parasite.

FIGURE 2. LIFE CYCLE OF *PLASMODIUM*, THE MALARIA PARASITE



Vector Ecology Profiles. Worldwide, about 70 species of *Anopheles* transmit malaria to man but, of these, only about 40 are important vectors.

General Bionomics. Female anopheline mosquitoes must ingest a bloodmeal for their eggs to develop. Feeding activity begins at dusk for many species, although many others feed only later at night or at dawn. Most anophelines feed on exposed legs, although some may feed on arms, ears or the neck. Infected females tend to feed intermittently and thus may bite several people. Eggs mature 3 to 4 days after the bloodmeal and are deposited one at a time, primarily in clean water with or without emergent vegetation, depending upon the mosquito species. A single female may deposit up to 300 eggs. Mosquito larvae feed on organic debris and minute organisms living in aquatic habitats. Oviposition sites include ground pools, stream pools, slow moving streams, animal footprints, artificial water vessels, and marshes. Deep water (over 1 m in depth) is generally unsuitable for larval development. There are 4 larval instars, and 1 to 2 weeks are usually required to reach the nonfeeding pupal stage. The pupa is active and remains in the water for several days to a week prior to adult emergence. The life span of females is usually only 1 to 3 weeks, although under ideal conditions female mosquitoes may live for 2 to 3 months. Longevity of individual species varies. A long life span is an important characteristic of a good vector. The older the anopheline population is in an endemic area, the greater the potential for transmission. Males live only a few days. Females mate within swarms of males, usually one female per swarm. Males and females feed on plant sugars and nectar to provide energy for flight and other activities.

Adult Feeding, Resting, and Flight Behavior. *Anopheles* spp. that are strongly attracted to humans are usually more important as vectors than those species that are strongly zoophilic. *Anopheles* generally fly only short distances from their breeding sites. The flight range is the distance traveled from the breeding site over the course of the mosquito's lifetime. This is important when determining how far from military cantonments or human settlements to conduct larviciding operations. Vectors that feed and rest indoors are more susceptible to control by residual insecticides.

Specific Bionomics:
There are a large number of malaria vectors in the region, especially India. In urban areas, *Anopheles stephensi* is the major vector. In most rural areas, *An. culicifacies* is the principal vector. In the foothills of northern and southern mountains, *An. fluviatilis* is the principal vector. Along the Bay of Bengal, *An. sundaicus* is the major vector. In Afghanistan and Pakistan, *An. superpictus* is an efficient vector in upland areas. Secondary vectors in lowland areas include *An. aconitus*, *An. annularis*, *An. subpictus*, *An.*

philippinensis, and *An. jeyporiensis*. In forested foothills and mountains, *An. dirus*, *An. minimus*, *An. maculatus* and *An. pulcherrimus* are vectors. Other significant vectors are *An. sergenti*, *An. hyrcanus* var. *nigerrimus*, *An. barbirostris*, *An. varuna*, and *An. umbrosus*.

1. Primary Vectors

Anopheles stephensi is a relatively small species that occurs widely in urban areas in India, Sri Lanka and Pakistan. It is highly anthropophilic and feeds primarily outdoors, generally before midnight. There are 2 cytologically different races, one urban and one rural. Both races occasionally feed indoors but rest indoors and outdoors. In urban areas, it is not a strong flier (< 2 km) and typically does not live past 6 gonotrophic cycles. In rural areas it often flies over 4.5 km. In many urban areas it occurs year-round. Larvae prefer relatively clean water and breed in cisterns, wells, undisturbed water puddles, and a variety of other manmade habitats.

Anopheles culicifacies occurs in Pakistan, Afghanistan, Nepal, India, Sri Lanka, and Bangladesh. At least 3 sibling species have been identified, with species A being the most important malaria vector. It is widely distributed except for the Kashmir region. In southern and central India it occurs year-round. The species has been found at elevations up to 2,000 m. It commonly occurs in houses and animal shelters. Adults rest in cracks and crevices and are zoophilic, readily feeding on cattle and occasionally on humans. *Anopheles culicifacies* feeds throughout the night, although feeding declines after 0300 hours. Indoor feeders readily remain indoors to rest, although *An. culicifacies* often feeds and rests outdoors. Larvae breed in clean, fresh water in irrigation channels, slow-moving streams, shallow tanks, borrow pits with grassy edges, and wells. Adults generally fly distances of 0.4 km or less but are known to fly 5.2 km from their habitat. This species is relatively long-lived and may survive through 8 gonotrophic cycles.

Anopheles fluviatilis is a vector in the northern and southern mountains. It commonly occurs in northwestern India, especially Baluchistan and Kashmir. It also occurs in Pakistan, Afghanistan, Nepal, Sri Lanka, and Bangladesh. At least 3 sibling species, designated S, T and U, have been identified in India. This species (larvae or adults) occurs year-round, except that it tends to be less abundant in the early part of the monsoon season. It occurs at altitudes up to 2,300 m. It feeds and rests indoors and outdoors, attacking man and animals through the night until 0300 hours. Larvae breed in stream beds, vegetated ponds, and edges of swamps. The flight range of adults usually does not exceed 0.5 km, but a range of 1.5 km has been recorded.

Anopheles sundaicus is a vector in India, Sri Lanka, and Bangladesh, primarily along the Bay of Bengal. It also occurs in the Ganges and Brahmaputra Deltas. Adults are strong fliers (often over 5 km) and feed on water buffalo and pigs in preference to humans. This species often feeds and rests indoors. Larvae breed in brackish water with a range of 0.4 to 3.0% salinity. The preferred salinity is 1.2 to 1.8%. Larvae are often found in fish ponds, especially if algal mats are present. *Anopheles sundaicus* is tolerant of pollution.

Anopheles superpictus is an important vector in Afghanistan and Pakistan, often occurring at high altitudes up to 2,100 m. This species is mainly zoophilic, feeding and resting outdoors. However, it also readily feeds on man. Larvae are often found in hilly streams and irrigation channels. River or stream beds covered with pebbles are ideal habitats. Sometimes, larvae live in association with *An. culicifacies* and *An. stephensi* in rural areas. Adult females are strong fliers, easily dispersing 5 km or more from their breeding sites.

2. Secondary Vectors

Anopheles aconitus is a small species about the size of *An. minimus*. This species occurs in eastern and southern India, Sri Lanka, Nepal, and Bangladesh. It occurs nearly year-round but is less abundant in the early monsoon season. It commonly occurs in houses and animal sheds and feeds on cattle as well as humans. It is a moderately strong flier (1.5 to 2.5 km). Larvae breed in rice fields, stream water drains containing grass, and other shaded pools and ponds with emergent vegetation. In Assam, northeastern India, *An. aconitus* breeds year-round.

Anopheles annularis is a brightly marked, minor vector species that occurs in parts of Assam and Orissa, as well as Nepal, Sri Lanka, and Bangladesh. There are 2 sibling species, designated as Species A and

Species B. Species B is considered the better vector. *Anopheles annularis* is highly zoophilic and prefers to feed and rest outdoors, primarily before midnight. This species occurs in large numbers during the post-monsoon seasons and into the dry, cool season. Larvae breed in rice fields or other permanent water bodies with emergent vegetation. Adults are moderately strong fliers that disperse up to 1.7 km or more from their breeding sites to find a host.

Anopheles dirus occurs in the forested foothills of northeast India, Bangladesh and Nepal. Known as the "white-kneed mosquito" because of markings between the tibia and tarsus, it comprises at least 3 sibling species within its South Asian geographic range. Although the complex is not common in most of this region, its members are efficient vectors because of their anthropophilic behavior and strong flight capability (2 to 4 km). *Anopheles dirus* feeds indoors and outdoors but rarely rests indoors, making it difficult to control with interior residual sprays. It usually feeds between 2200 hours and 0400 hours. Larvae are most often found in isolated stream pools, undisturbed puddles, and occasionally in cisterns or water storage jars.

Anopheles jeyporiensis is a small species resembling *An. minimus*. Its distribution in the region is largely eastern and southern India, Nepal, and Bangladesh. It frequently occurs in houses and cattle sheds. Larvae breed in and around rice fields, seepages from rice fields, grassy streams, and edges of ponds. It may occur at altitudes up to 1,000 to 2,000 m. Its flight range often exceeds 0.8 km.

Anopheles maculatus occurs in hilly areas of India, Nepal, Sri Lanka and Bangladesh. This species is primarily zoophilic, but there are 3 sibling species, and one of these is more anthropophilic. *Anopheles maculatus* may feed indoors or outdoors but rests outdoors after feeding. It feeds primarily from 1800 to 2100 hours. It disperses at least 1 km from its breeding sites. Larvae breed in sunlit stream margins, edges of ponds, ditches and rice fields.

Anopheles minimus is a small mosquito that occurs in northeastern foothills of the Himalayas in India, in Sri Lanka, in Bangladesh, in Afghanistan and Pakistan. It is a moderately strong flier (up to ~3 km), feeds strongly on humans indoors, but will feed outdoors on man and animals. It generally rests indoors. This species occurs year-round in many areas. The larvae breed in slow-moving stream margins in the dry season, but also occur in ground pools in the rainy season. Other habitats include irrigation ditches, springs, seepages, and rice fields.

Anopheles philippinensis is a small species that occurs in the plains of West Bengal, in northeastern India, Nepal, and Bangladesh. It is most common during the monsoon and post-monsoon seasons. It is largely zoophilic and feeds on animals or man, resting outdoors and, occasionally, indoors. It generally does not disperse more than 1 km from its breeding habitats. Larvae breed in rice fields and other brightly sunlit permanent bodies of clean water with submerged vegetation.

Anopheles pulcherrimus occurs in Afghanistan and may be an important vector there. It prefers feeding on cattle outdoors but may feed on man as well. This species rests indoors and outdoors. It feeds primarily before midnight, but feeding continues throughout the night. It is considered a strong flier, with a flight range of 5 km or more. Larvae develop in streams, stream pools, rice fields, and irrigation plots.

Anopheles sergentii is a small species that is narrowly distributed in northwest India, Afghanistan, and Pakistan. This species feeds on man or animals indoors or outdoors, and rests in shelters or houses after feeding. *Anopheles sergentii* is a strong flier that may travel more than 5 km. Larvae breed in irrigation plots, rice fields, or other sunlit pools with emergent vegetation.

Anopheles subpictus occurs in Afghanistan, Pakistan, Sri Lanka, Nepal, and India. There are 2 sibling species (A and B) that feed mainly on animals outdoors but also on humans indoors and outdoors. In southern India and Sri Lanka, it occurs year-round. *Anopheles subpictus* feeds indoors, primarily from 1800 to 2100 hours, and is rarely found biting indoors after 2300 hours. There is a second, predawn feeding peak. It occurs in large numbers during the late monsoon and post-monsoon seasons, with a secondary peak in the spring after the start of the spring rains. Larvae can be found in muddy pools near houses but also inhabit gutters, borrow pits, and brackish waters. Species A prefers water with low salinity, while species B prefers brackish water. Both species prefer breeding sites with emergent vegetation or

algae. *Anopheles subpictus* is a moderately strong flier, with a range of 3.2 km or more. With its coastal distribution, Species B is considered to be the better vector. In northern parts of the region, *An. subpictus* is most abundant during the pre-monsoon through post-monsoon seasons (June to November).

3. Miscellaneous Vectors
Anopheles barbirostris is a large, dark mosquito that occurs widely in the region, including India and Nepal. This species occurs in the monsoon and post-monsoon seasons. It is primarily zoophilic but will feed on man. It often bites during the early daylight hours or when the sun is hidden and will feed during cloudy days. It attacks man and animals indoors and outdoors and may rest indoors. Larvae often breed in swamps or ponds but also occur in shaded rice fields, ditches, earthen wells, and sometimes brackish water. This species is a moderately strong flier but does not have a long lifespan.

Anopheles hyrcanus var. *nigerrimus* is a large, dark mosquito that occurs in northeastern India, Sri Lanka, Afghanistan, Pakistan, Nepal, and Bangladesh. This species feeds readily on humans and animals early in the evening, and occasionally in shaded areas during the day. It feeds and rests outdoors. Larvae are found in deep ponds and ditches, borrow pits, rice fields, and swamps. This species' flight range is unknown. It generally does not have a long lifespan but is considered an important vector in Afghanistan.

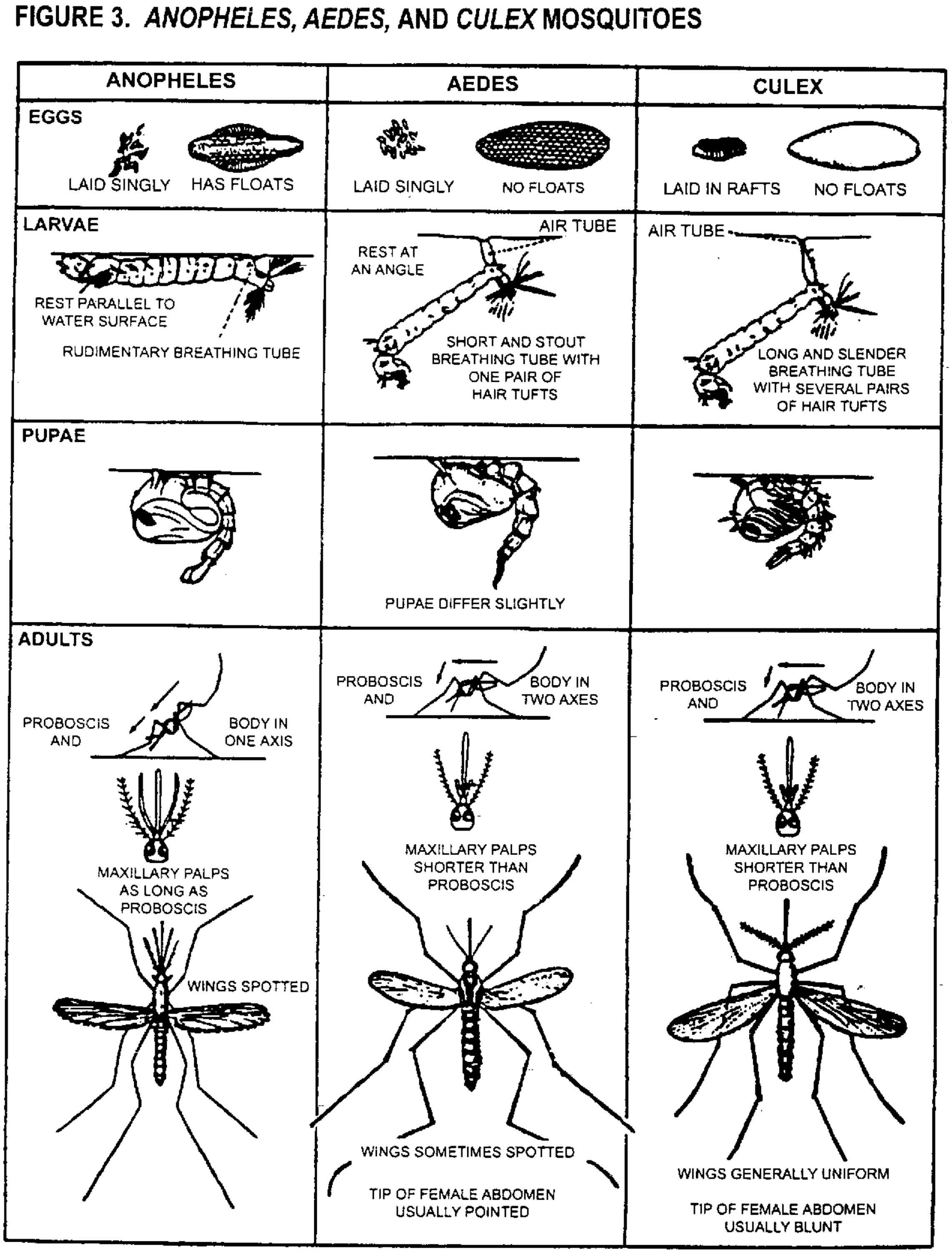
Anopheles umbrosus is a large mosquito that occurs in India. It primarily feeds on animals and only occasionally bites man. It feeds and rests outdoors, seeking hosts early in the evening and again after dawn in the morning. Larvae breed in stagnant water, stream pools, pond edges, ditches, and rice fields. The adult flight range is unknown.

Anopheles varuna is a small mosquito similar in appearance to *An. minimus*. It occurs in the foothills of the Eastern Ghat Mountains, central and northern India, Nepal, and Sri Lanka. It is most abundant during the late monsoon and post-monsoon seasons. It bites man and animals indoors and outdoors, and frequently rests indoors. It has a short flight range, dispersing approximately 1 km or less. Larvae occur in stagnant shaded or unshaded water in ditches, domestic wells, ground pools, and stream margins.

Vector Surveillance and Suppression. Light traps are used to collect night-biting mosquitoes, but not all *Anopheles* spp. are attracted to light. The addition of the attractant carbon dioxide to light traps increases the number of species collected. Traps baited with animals, or even humans, are useful for determining feeding preferences of mosquitoes collected (use of humans as bait may be subject to the requirements of human-use protocols). Adults are often collected from indoor and outdoor resting sites using a mechanical aspirator and flashlight. Systematic larval sampling with a long-handled white dipper provides information on species composition and population dynamics that can be used to plan control measures.

Anopheles mosquitoes have unique morphological and behavioral characteristics that distinguish them from all other genera of mosquitoes (Figure 3). Adult *Anopheles* feed on the host with the body nearly perpendicular to the skin. Other genera of mosquitoes feed with the body parallel or at a slight angle to the skin. These characteristics can easily be used by inexperienced personnel to determine if *Anopheles* are present in an area.

Figure 3. ***Anopheles, Aedes,*** **and** ***Culex*** **Mosquitoes.**



Eggs are laid singly and float on the water surface. They are dark, about 1 mm in length, and in most species are boat-shaped with a pair of lateral floats. The shape, size and pattern of the floats can be used to distinguish closely related species.

Anopheles larvae hang with the body parallel to the water surface by means of specialized palmate hairs that are unique to the genus. *Anopheles* are also the only mosquitoes that lack an air siphon. *Anopheles* larvae feed on micro-organisms and small particles floating on the water surface. Entomologists have exploited this feeding behavior to control *Anopheles* larvae by dispersing insecticidal dusts that stay on the water surface. Larvae are easily disturbed by shadows or vibrations and respond by swimming quickly to the bottom. They may wait a few seconds or even minutes before they resurface. This behavior should be taken into consideration when surveying for mosquito larvae.

Malaria suppression includes elimination of gametocytes from the bloodstream of the human reservoir population, reduction of larval and adult *Anopheles* mosquito populations, use of **personal protective measures** such as skin repellents, permethrin-impregnated uniforms and bednets to prevent mosquito bites, and chemoprophylaxis to prevent infection. Specific recommendations for chemoprophylaxis depend on the spectrum of drug resistance in the area of deployment. Command enforcement of chemoprophylactic measures cannot be overemphasized. When Sir William Slim, British Field Marshal in Southeast Asia during World War II, strictly enforced chemoprophylactic compliance by relieving inattentive officers, malaria attack rates declined dramatically. During the Vietnam War, malaria attack rates dropped rapidly in military personnel when urine tests were introduced to determine if chloroquine and primaquine were being taken.

Many prophylactic drugs, such as chloroquine, kill only the erythrocytic stages of malaria and are ineffective against the latent hepatic stage of *Plasmodium* that is responsible for relapses. Therefore, even soldiers who take chloroquine appropriately during deployment can become infected. Individuals who are noncompliant with the prescribed period of terminal prophylaxis are at risk for late relapses upon their return to the United States. During the Vietnam War, 70% of returning troops failed to complete their recommended terminal prophylaxis. The majority of cases in military personnel returning from Operation Restore Hope in Somalia resulted from failure to take proper terminal prophylaxis.

Application of residual insecticides to the interior walls of buildings and sleeping quarters is an effective method of interrupting malaria transmission when local vectors feed and rest indoors. Nightly dispersal of ultra low volume (ULV) aerosols can reduce exophilic mosquito populations. Larvicides and biological controls (e.g., larvivorous fish) can reduce populations of larvae at their aquatic breeding sites before adults emerge and disperse. Insecticides labeled for mosquito control are listed in TIM 24, Contingency Pest Management Pocket Guide. Chemical control may be difficult to achieve in some areas. After decades of malaria control, many vector populations are now resistant to insecticides (Appendix C. Pesticide Resistance in South Central Asia). Specially formulated larviciding oils can be used to control insecticide-resistant larvae. Pathogens, such as *Bacillus thuringiensis israelensis* and *B. sphaericus*, and insect growth regulators have also been used to control resistant larvae. However, there is growing evidence that resistance to these control agents has developed, although it is not nearly as widespread as resistance to chemical insecticides.

Sanitary improvements, such as filling and draining areas of impounded water to eliminate breeding habitats, should be undertaken to the extent possible. Instead of draining marshy areas, they can be excavated to form deep permanent impoundments with well-defined vertical banks that are unsuitable habitat for mosquito larvae. Other methods of source reduction can be utilized. In the Indian Subcontinent, water tanks are commonly sited on rooftops and are important breeding places for *Anopheles stephensi*, the vector of urban malaria. Fitting these with mosquito screening can prevent breeding unless the screens become torn or are removed.

The proper use of repellents and other **personal protective measures** is thoroughly discussed in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance. The use of bednets impregnated with a synthetic pyrethroid, preferably permethrin, is an extremely effective method of protecting sleeping individuals from mosquito bites. Buildings and sleeping quarters should be screened to prevent entry of mosquitoes and other blood-sucking insects. The interior walls of tents and bunkers can be treated with permethrin to control resting vectors. See Appendix F for further information on **personal protective measures**.

B. Dengue Fever.

Dengue (aka: Breakbone fever; Dandy fever) is an acute febrile disease characterized by sudden onset, fever for 3 to 5 days, intense headache, and muscle and joint pain. It is commonly called "breakbone fever" because of the severity of pain. There is virtually no mortality in classical dengue. Recovery is complete, but weakness and depression may last several weeks. Dengue is caused by a *Flavivirus* and includes 4 distinct serotypes (dengue 1, 2, 3 and 4). Recovery from infection with 1 serotype provides lifelong immunity from the same serotype but does not confer protection against other serotypes. Dengue hemorrhagic fever (DHF) and associated dengue shock syndrome (DSS) were first recognized during a 1954 epidemic in Bangkok, Thailand. DHF/DSS have spread throughout Southeast Asia, Indonesia and the southwest Pacific, Latin America and the Caribbean. DHF requires exposure to 2 serotypes, either sequentially or during a single epidemic involving more than one serotype. DHF is a severe disease that produces high mortality in children.

Military Impact and Historical Perspective. Dengue epidemics were reported in 1779 and 1780 in Asia, Africa and North America. For the next 150 years there were usually long intervals between major epidemics (20 to 40 years), mainly because the viruses and their mosquito vector were transported between population centers by sailing vessels. Dengue virus was first isolated and characterized in the 1940s. A global pandemic of dengue began in Southeast Asia after World War II and has intensified during the last 15 years. Epidemics of dengue are noted for affecting large numbers of civilians or military forces operating in an endemic area. Outbreaks involving 500,000 to 2 million cases have occurred in many parts of the world. During World War II, the incidence of dengue was largely restricted to the Pacific and Asiatic theaters. Only scattered cases of dengue were reported from other theaters, including North Africa. Campaigns in the Pacific were marked by dengue epidemics, and throughout the war the US Army experienced nearly 110,000 cases. At Espiritu Santo in the Pacific, an estimated 25% of US military personnel became ill with dengue, causing a loss of 80,000 man-days. Although dengue was endemic in most of the China-Burma-India theater, the majority of cases among U.S. troops occurred in the vicinity of Calcutta, India. From 1942 to 1944, the incidence of dengue was 25 cases per 1,000 per annum. Dengue was an important cause of febrile illness among U.S. troops during Operation Restore Hope in Somalia.

Disease Distribution. Dengue is the most important mosquito-borne virus affecting humans and is present in nearly all tropical countries. Its distribution is congruent with that of its primary vector, *Aedes aegypti*, between 40^{o} N and 40^{o} S latitude. In recent years dengue, especially DHF, has been expanding throughout the world. An estimated 2.5 billion people live in areas at risk for dengue transmission, and 30 to 50 million cases of dengue are reported annually.

Epidemics as well as sporadic cases occur year-round in most urban and semi-rural areas of South Central Asia below 1,000 m. Epidemics generally coincide with the monsoon rainy season when mosquito populations are high. All 4 dengue serotypes are circulating in South Central Asia. Dengue is endemic countrywide in Bangladesh but occurs primarily in the southern plains region of Bhutan. During August 2000, there were 3 reports of DHF/DSS in travelers returning to the U.S. from Bangladesh. The magnitude of the dengue outbreak in Bangladesh remains unknown, but DHF/DSS has not been previously reported in Bangladesh.

Dengue outbreaks occur annually in India, with most outbreaks occurring in the northcentral states. Relatively few cases are reported from the western states. In southern states the risk of dengue is year-round, but dengue transmission occurs in northern India primarily from April through November. In metropolitan Calcutta, DHF was first recorded during 1963, and another outbreak of DHF occurred there in 1990. Subsequent outbreaks of dengue fever occurred in cities on the east coast of India as well as in central and northern parts of the country, including an epidemic in Madras during 1989 and 1990. The most recent and largest epidemic of DHF/DSS occurred in Delhi, India, and adjoining areas, from August through November 1996. A total of 8,900 cases were reported, with a death rate of 4.2%. Delhi, situated in the northern part of India, experienced outbreaks of dengue due to different viral serotypes in 1967, 1970, 1982 and 1988, but no confirmed cases of DHF/DSS were reported during these epidemics. The 1988 epidemic affected about 30% of the inhabitants of the city. All outbreaks in Delhi occurred during the monsoon rainy season (August to November) and subsided with the onset of winter. However, an outbreak

of dengue fever occurred in Jalore town, southwest Rajasthan, in April and May 1985. This was the first reported outbreak of dengue in the arid zone of western Rajasthan; it occurred in the dry season, in contrast to other parts of India where dengue outbreaks follow heavy rains.

Dengue has not been reported from Nepal in recent years. Risk of transmission occurs at lower elevations, but the risk is low. In Pakistan the risk of transmission is highest in southeastern areas. The first Pakistani outbreak of DHF occurred in Karachi and began in August 1994 following a period of unusually heavy rains. The magnitude of the Karachi epidemic was never fully known, but the number of patients seen by physicians was estimated to be in the thousands. The risk of dengue is countrywide in Sri Lanka, although elevated in urban areas. Rapid urbanization resulting in slums and shantytowns has increased breeding habitats for dengue vectors and the potential for epidemic transmission of dengue virus in many areas of South Central Asia..

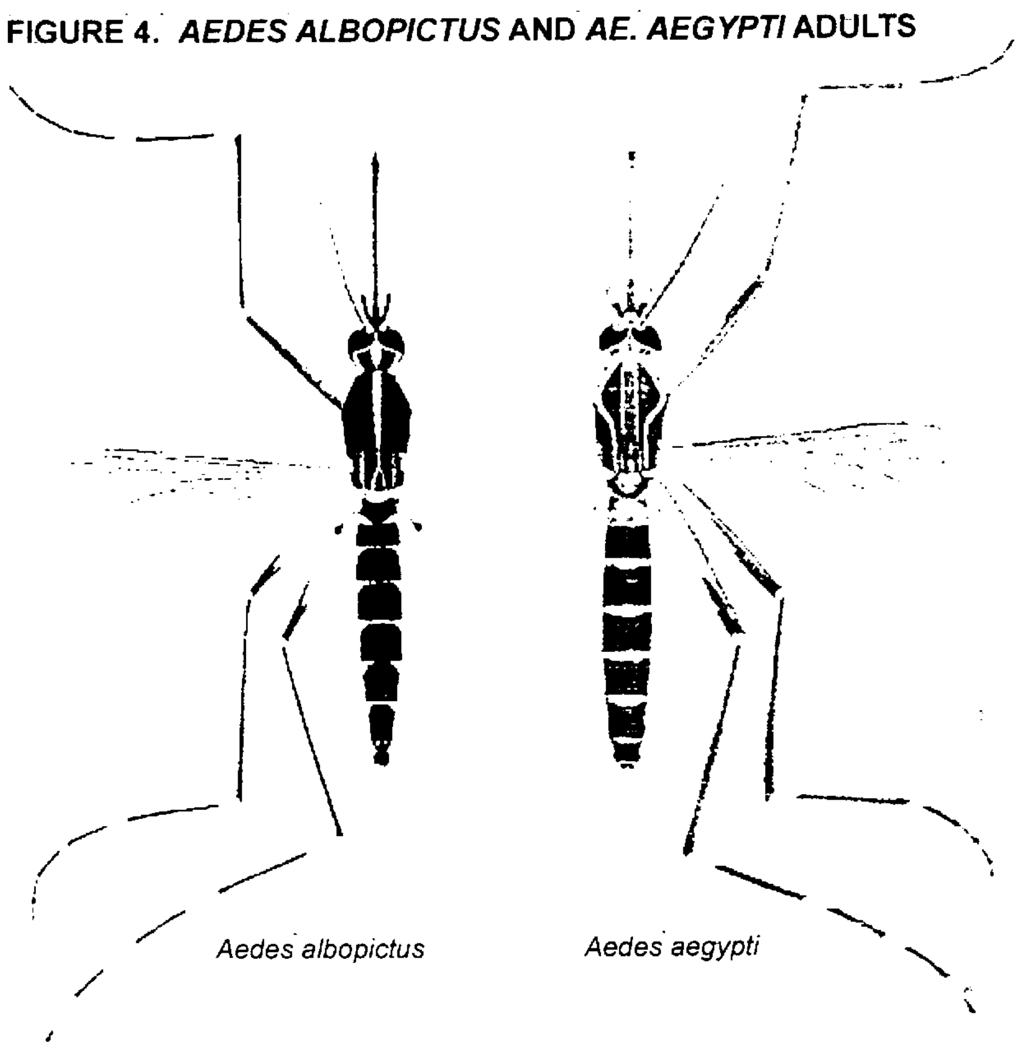
Transmission Cycle(s). Dengue virus is primarily associated with *Aedes* mosquitoes in the subgenus *Stegomyia*. The virus is maintained in a human-*Ae. aegypti* cycle in tropical urban areas. A monkey-mosquito cycle serves to maintain the virus in sylvatic situations in Southeast Asia and West Africa. An epizootic of dengue was detected among toque macaques, *Macaca sinica*, at Polonnaruwa, Sri Lanka, between October 1986 and February 1987. The epizootic was highly focal, but transmission among macaques in the wild may have important public health implications for the region. Mosquitoes are able to transmit dengue virus 8 to 10 days after an infective blood meal and can transmit the virus for life. Dengue virus replicates rapidly in the mosquito at temperatures above 25 ^{o}C. Evidence for transovarial transmission of dengue virus in *Ae. aegypti* has been found in India.

Vector Ecology Profiles. *Aedes aegypti*, the primary vector of dengue, is widespread throughout the region, although it has not been reported from Nepal and Bhutan and has a restricted distribution in Afghanistan, where it occurs only in limited lowland areas. This species is more common in cities or in villages than in rural areas. It is very abundant in slums and shantytowns, where drinking water is stored in tanks or jars and there are numerous artificial containers. Rapid and uncontrolled urbanization in Pakistan, India and Sri Lanka has greatly increased the abundance of this mosquito. *Aedes aegypti* deposits its eggs singly or in small groups of 2 to 20 above the water line of its habitat. Eggs may withstand dessication for 3 months or more. Larvae emerge after eggs have been submerged for 4 or more hours. *Aedes aegypti* larvae live in artificial water containers, including flowerpots, cisterns, water jugs, and tires. Larvae were found in 20% of the wells in the villages of Gori and Singarthope on a small island near Cuddalore, Tamil Nadu State. Masonry water tanks were found to be the preferred breeding site during the 1990 dengue epidemic in Calcutta. Water is stored in a variety of containers in most Indian homes due to irregular water supply. In some surveys, indoor containers of all types had *Ae. aegypti* larvae more often than containers outdoors. The abundance of larval populations usually parallels fluctuations in both rainfall and humidity. Surveys were conducted during 1997 in used and waste tire dumps along the national highways and trunk roads of Assam, Arunachal Pradesh, Meghalaya and Nagaland, India. Thirty to 88% of tires contained larvae of *Ae. aegypti* or *Ae. albopictus*, although *Ae. aegypti* predominated. The study confirmed the importance of waste tire dumps as breeding sites for dengue vectors. Occasionally, *Ae. aegypti* is reported from coconut shells or bamboo stumps, although these are more typical habitats for *Ae. albopictus*. Larvae prefer relatively clean and clear water. They develop quickly in warm water, maturing to the pupal stage in about 9 days. Pupae remain active in the water container until adult emergence, 1 to 5 days after pupation. *Aedes aegypti* rarely disperses more than 50 m from its breeding site, but over several days it can disperse as far as 500 to 600 m. It does not fly when winds exceed 5 km per hour.

Aedes aegypti prefers human hosts and feeds primarily around human habitations. It is a diurnal feeder and readily enters homes. This species is not attracted to light; rather, it responds to contrasting light and dark areas presented by human dwellings. When feeding outdoors, it prefers shaded areas. It feeds on the lower legs and ankles, increasing its biting activity when temperatures and humidity are high. It is easily disturbed when feeding and, because it feeds during the day, is often interrupted by the movements of its host. This behavior results in multiple bloodmeals, often taken within the same dwelling, which increases transmission of virus. *Aedes aegypti* rests in cool, shaded areas within dwellings, often in closets, under tables, or in sheds. Similarly, it rests outdoors in shaded areas of trees, shrubs, and structures.

Aedes albopictus is second only to *Ae. aegypti* in importance as a vector of dengue. Adults of the two species can easily be distinguished by the pattern of silver scales on the top of the thorax (Figure 4). *Aedes albopictus* is more common in rural than urban areas. *Aedes albopictus* has larval and adult feeding habits similar to *Ae. aegypti* but is more commonly found breeding in natural containers, such as tree holes, leaf axils, and fallen fruit husks. It is a slightly stronger flier than *Ae. aegypti*. *Aedes albopictus* is strongly anthropophilic but has a broader host range than *Ae. aegypti* and may feed on oxen, dogs and pigs. *Aedes albopictus* does not readily feed on birds.

Figure 4. Aedes albopictus and Aedes aegypti adults.



Aedes albopictus most often occurs in rural, partially forested areas, particularly along forest fringes, where it may be present in large numbers. It is generally absent from the interiors of deep forests or jungles. Eggs are deposited singly or in small numbers above the water line and hatch after being flooded for 1 to 7 days, depending on the state of embryo development within the egg. They may withstand desiccation for several months if not flooded, and they are the overwintering stage in northern parts of the region. Larvae occur in manmade containers, bamboo stumps, coconut shells, tires, treeholes, and rock pools. These containers are usually slightly shaded. The larval development

time varies from 5 days to 3 weeks, depending on temperature. Adults emerge 1 to 5 days after pupation. Females feed every 3 to 5 days for the duration of their life, which lasts from 1 to 4 weeks. Females fly close to the ground and generally not further than 100 m from their breeding sites. They do not fly in winds over several km per hour. Adults may also feed on nectar from plants. Autogeny occurs in this species, although usually only 2 to 4 eggs are produced in this manner. Peak feeding periods outdoors are generally early morning and late afternoon. Adults usually feed outdoors and rest outdoors in undergrowth. However, indoor feeding and resting behavior also occurs. *Aedes albopictus* is most abundant during the monsoon and post-monsoon months of June through November, with greatly reduced numbers occurring in the dry season.

Vector Surveillance and Suppression. Landing rate counts provide a quick relative index of adult abundance. The number of mosquitoes that land on an individual within a short period of time, usually 1 minute, is recorded. Resting collections consist of the systematic search for dengue vectors in secluded places indoors, such as in closets and under furniture. Resting collection studies performed with mechanical aspirators are an efficient but labor-intensive means of evaluating adult densities. Densities are recorded either as the number of adult mosquitoes per house or the number of adult mosquitoes collected per unit of time.

Several indices have been devised to provide a relative measure of the larval populations of *Ae. aegypti*. The house index is the percentage of residences surveyed that have containers with larvae. The container index is the percentage of containers at each premise that have larvae. The Breteau index is more widely used and is the number of positive containers per 100 premises. There is a risk of dengue transmission when the Breteau index goes above 5, and emergency vector control is indicated when the index exceeds 100. During a dengue outbreak in a typical rural area of Haryana State in northern India, the house index for *Ae. aegypti* was 75%, the container index was 19%, and the Breteau index was 121. Adult egg-laying activity can be monitored using black oviposition traps that container-breeding *Aedes* readily utilize. The number of eggs laid in the ovitrap gives a relative indication of abundance of dengue vectors. Ovitraps are especially useful for the early detection of new infestations in areas from which dengue vectors had been eliminated.

No vaccine and no specific treatment exists for dengue, so control of dengue fever is contingent upon reducing or eliminating vector populations. Ground or aerial applications of insecticidal aerosols have been relied upon to reduce adult populations during epidemics of dengue. Many vector control specialists have questioned the efficacy of ULV adulticiding. In some outbreaks of dengue fever, ULV dispersal of insecticides has had only a modest impact on adult mosquito populations. *Aedes aegypti* is a domestic mosquito that frequently rests and feeds indoors and therefore is not readily exposed to aerosols. The sides of large storage containers should be scrubbed to remove eggs when water levels are low. Water should be stored in containers with tight-fitting lids to prevent access by mosquitoes. A layer of oil will prevent mosquito eggs from hatching and will kill the larvae. The elimination of breeding sources, such as old tires, flowerpots, and other artificial containers, is the most effective way to reduce mosquito populations and prevent dengue outbreaks. In Singapore, passage of sanitation laws and their strict enforcement to eliminate breeding sites reduced the house index for *Ae. aegypti* larvae from 25% to 1%. Proper disposal of trash, bottles and cans at military cantonments must be rigidly enforced. The individual soldier can best prevent infection by using **personal protective measures** during the day when vector mosquitoes are active. Wear permethrin-impregnated BDUs and use extended-duration DEET repellent on exposed skin surfaces (see TIM 36).

C. Japanese Encephalitis.

Japanese encephalitis (JE) is caused by a *Flavivirus* in the family Flaviviridae and is closely related to St. Louis encephalitis virus. Many infections are inapparent or produce a mild systemic illness characterized by fever, headache or asceptic meningitis. Serological studies indicate a ratio of inapparent to apparent infections as high as 300 to one. The incubation period is 5 to 15 days. Severe infections are marked by acute onset, high fever, severe headache, and vomiting. Inflammation of the brain, spinal cord and meninges can cause stupor, tremors, convulsions (especially in infants), spastic paralysis, coma and death. Case fatality rates can be as high as 60%. Fatal cases result in coma and death within 10 days. In past

epidemics, approximately 25% of clinical cases are rapidly fatal, 50% lead to permanent neurological or psychiatric sequelae, and 25% resolve within 1 to 2 weeks. Neurological impairment is most severe in infants. No specific therapy is effective and treatment is primarily supportive.

Military Impact and Historical Perspective. Although reports of a disease resembling JE go back to 1870 in Japan, the disease attracted little attention until a large epidemic in 1924 that resulted in 6,125 cases and 3,797 deaths. JE was recognized in Korea in 1926 and in China in 1940. Japanese encephalitis virus (JEV) was first isolated in 1935 from the brain tissue of a fatal encephalitis case in Japan. No arthropod-borne virus causing encephalitis had been discovered, nor had the diseases that they cause been recognized prior to the 1930s. Consequently, there is nothing about JE or other arthropod-borne encephalitides in the medical history of World War I. During World War II, there was considerable preparation by U.S. military medical personnel for arthropod-borne viral encephalitides, but this group of diseases turned out to be less of a threat than anticipated. There was one small but important outbreak of Japanese encephalitis that involved both natives and troops on the island of Okinawa in the summer of 1945. Only 11 cases with two deaths were confirmed in U.S. military personnel. However, large numbers of U.S. troops had been assembled for the projected invasion of the home islands of Japan, and the danger of an epidemic was of great concern to medical personnel. The news of “a dread Japanese brain disease” on Okinawa had also spread among the troops, with demoralizing effects. The commercial availability of effective vaccines against JE has greatly reduced the threat of this crippling disease to future military operations in JE-endemic areas. Only about 1 case of JE is reported per year in U.S. civilian or military personnel traveling or living in Asia.

Disease Distribution. JE is endemic in at least 21 countries from the maritime provinces of Russia, North Korea and Japan southward through China, Southeast Asia and Indonesia, and westward through the Indian Subcontinent. It is the leading cause of viral encephalitis in Asia, with 30,000 to 50,000 cases annually. JE was recognized in India in 1954. Sporadic cases were confined primarily to southern areas of the country until 1978, when severe epidemics erupted in the northeastern states of Bihar, Uttar Pradesh and West Bengal. Simultaneously, epidemic JE first appeared in the Terrai region of Nepal. Currently, JE cases are reported from all states in India except Arunachal, Dadra, Daman, Diu, Gujarat, Himachal, Jammu, Kashmir, Kerala, Lakshadweep, Meghalaya, Nagar Haveli, Orissa, Punjab, Rajasthan, and Sikkim. Most JE cases are recorded in the eastern and northeastern states, where outbreaks occur annually, often involving several thousand cases. Only sporadic cases occur in southern states, except for the southwestern state of Kerala. In the fall of 1999, an outbreak occurred in Andhra Pradesh State that involved 965 cases with 200 deaths. Risk of transmission is low in western states. The transmission season in southern India is: 1) May through October in Goa, 2) October through January in Tamil Nadu, 3) August through December in Karnataka, with a second peak from April through June in the Mandya District, and 4) September through December in Andhra Pradesh. In northern India the period of highest transmission is normally July through December.

The risk of JE in Afghanistan is low. Historically, cases have occurred along the eastern borders. JE had not been reported from Bangladesh until 1977, when an outbreak began in the Modhupur Forest area of central Tangail District. JE is now widespread in the country and sporadic cases occur every year, but the last serious outbreak was the 1977 epidemic. Risk of transmission is greatest during the monsoon season from June through November. There are no data for Bhutan, but JE is believed to occur in the southern foothill districts. Risk of transmission is highest during the rainy season from July through September.

JE is highly endemic in the Terai plain and inner Terai zone of Nepal. Cases occur throughout the year and usually increase between June and October, especially in the southern agricultural areas bordering India. Incidence and prevalence seem to be increasing as deforestation and extensive cultivation create larger areas with ecological conditions favorable for JE. In 1995, 772 cases of JE were reported, with 126 deaths. Over 1,000 cases had been reported in Nepal through April 2000. Epidemics have occurred every year since 1990 in the area around Tikapur in southwestern Nepal. Between July and October 1997, 339 patients were admitted to the Tikapur hospital with JE. A limited serosurvey in the Kathmandu Valley found that 50% of the pigs had JEV antibodies, and human cases occurred for the first time in the area in 1995. Transmission of JE normally occurs below 1,000 m, but the elevation of the Kathmandu Valley is 1,300 m.

The risk of JE in Pakistan is low. A few cases have been reported near Karachi, and transmission may occur in the central deltas. Risk of transmission is elevated from June through January. JE is endemic in all but mountainous areas of Sri Lanka and is periodically epidemic in the northern and central provinces. Peak transmission occurs from October through January, with a secondary peak of May through June. JE has been increasing due to the Mahaweli Development Program. This is an ambitious project involving irrigation, agriculture and hydropower generation. Based in Sri Lanka's dry zone, it covers 39% of the whole island. Populations of *Culex tritaeniorhynchus* have greatly increased, as has the incidence of encephalitis. Defects in construction and subsequent deficiencies in operation and maintenance of irrigation schemes create vector breeding habitats during water delivery and drainage. Large outbreaks of JE have occurred around Anuradhapura, Sri Lanka. The distribution of Japanese encephalitis is depicted in Figure 5.

Figure 5. Distribution of Japanese Encephalitis in South Central Asia.

FIG. 5. DISTRIBUTION OF JAPANESE ENCEPHALITIS IN SOUTH CENTRAL ASIA (DARK SHADING).

Transmission Cycle(s). JE is maintained in essentially rural zoonotic cycle between rice field breeding mosquitoes and water birds such as egrets, herons and ibises. Pigs are important reservoir hosts that develop high viremias and, when abundant, serve as the primary amplifying host for JEV. Mass rearing of pigs is not as common in the Indian Subcontinent as in other parts of Asia. Cattle are more abundant than swine. Bovines develop little or no viremia but do serve as a source of blood meals that help produce high vector populations. Rodents and other domestic animals are not important in natural cycles of JEV transmission. Almost all domestic animals can be infected by JEV. Adult animals rarely develop signs of illness, although fatal encephalitis does occur in horses. Several species of bats are susceptible to JEV and develop viremias for 6 days or more that are sufficient to infect mosquitoes. Human infections are usually a consequence of increased vector densities associated with increased rainfall or irrigation.

Vector Ecology Profiles. Primary vectors in South Central Asia are *Cx. tritaeniorhynchus*, *Cx. fuscocephala*, *Cx. gelidus*, *Cx. pseudovishnui*, and *Cx. vishnui*. Additional vectors include *Cx. pipiens quinquefasciatus* (*Cx. pipiens fatigans*), *Cx. whitmorei*, *Cx. bitaeniorhynchus*, *Mansonia annulifera*, *Ma. uniformis*, *Anopheles subpictus*, and *Anopheles hyrcanus* s.l. Of the primary vector species, *Cx. tritaeniorhynchus* is considered to be the most important vector of JEV in most of the region.

Culex tritaeniorhynchus is the most common JE vector in South Central Asia. It feeds readily on the amplifying hosts, pigs and ardeid birds, as well as humans. It also is strongly attracted to cattle. This species is both endophagic and exophagic, and is endophilic, especially during the cool months in northern parts of the region. Adults begin feeding early in the evening and continue feeding throughout the night, with decreasing activity after 0200 hours. They are moderately strong fliers that will travel an average of 3 km for a blood meal. *Culex tritaeniohynchus* deposits egg rafts on the water surface that contain about 75 to150 eggs each. Rafts are deposited 3 to 4 days following a bloodmeal. Eggs hatch 2 to 4 days after deposition. Larval development requires 7 to 9 days at a temperature range of 25 to 30 ^{o}C. At lower temperatures, larval development may require 15 to 20 days. The pupal stage lasts about 2 days. Adult populations display 2 population peaks in the northern regions, during the pre-monsoon periods of May through June, and again in the post-monsoon period from November through March. Adults are entirely absent from late November to early February in northern regions, and these cool months are spent in the larval stage. In southern areas, peak abundance occurs from May through June, and again from October through December. Common oviposition sites include rice fields, water troughs, irrigation spillovers, and undisturbed ground pools. Rice fields are generally colonized soon after the planting and flooding of the paddy. Larvae of *Cx. tritaeniorhynchus* generally prefer lightly shaded ground pools with low concentrations of organic matter and some emergent vegetation. Paddy fields are ideal breeding sites for this species. Increased irrigation and expansion of rice growing in the region has increased the abundance of *Cx. tritaeniorhynchus* and the potential for transmission of JEV to the human population. It has been calculated that an average paddy plot (320 sq m) can produce 30,000 adults daily. Transovarial transmission has been demonstrated in this species, although its importance in maintaining the virus is unknown.

Culex vishnui and *Cx. pseudovishnui* occur widely in most of the region. Their seasonal abundance is similar to *Cx. tritaeniorhynchus*, with a peak in the pre-monsoon to early monsoon season and another peak in the post-monsoon season. Their breeding sites are primarily paddy fields and shaded ground pools. These species are chiefly exophagic and exophilic, preferring to rest in fields or forests, although a small percentage will feed on man in houses. *Culex vishnui* feeds primarily on pigs, while *Cx. pseudovishnui* is more attracted to cattle. Birds are also fed upon, especially by *Cx. pseudovishnui*. The life cycle is similar to *Cx. tritaeniorhynchus*, but the population peaks generally occur from March through April and September through October. The flight range of these species is also similar to *Cx. tritaeniorhynchus*, averaging about 3 km from the breeding site to feeding sites. Transovarial transmission has been demonstrated in *Cx. pseudovishnui*.

Culex fuscocephala occurs widely throughout the region. Its life cycle and seasonal abundance are similar to *Cx. tritaeniorhynchus*. Seasonal peaks occur from March to April, and again from August to September. Larval breeding sites include rice fields, borrow pits, and temporary ground pools with low organic content and some shade. This species is mildly endophagic, but feeds readily outdoors. It feeds continuously throughout the night except just before dawn, when feeding decreases sharply. *Culex fuscocephala* is

largely zoophilic, preferring cattle to other hosts. However, it also feeds occasionally on pigs, birds and humans. This species rarely disperses more than 2 km from its breeding sites.

Culex gelidus has a life cycle and seasonal abundance similar to *Cx. vishnui* and *Cx. pseudovishnui*. This species is strongly attracted to pigs. It also feeds readily on cattle and humans. Peak abundance occurs during the monsoon seasons when borrow pits, ditches, rice fields and ground pools are widely available as larval habitats. Larvae frequently breed in pits used to soak coconut husks to make rope and mat fibers. Although its densities are often low in central and northern India, *Cx. gelidus* is abundant in southwestern India and Sri Lanka. It is both endophilic and exophilic and readily feeds indoors and outdoors. Its life cycle is similar to that of *Cx. tritaeniorhynchus*. However, its average dispersal from breeding sites is less than 2 km.

Culex p. quinquefasciatus is a common mosquito throughout the region. This species is both endophagic and exophagic, and is relatively endophilic, especially during the cool months in northern parts of the region. Adults feed early in the evening, usually within two hours of sunset, and are strong fliers that will travel 3 to 5 km for a blood meal. Details on the bionomics of *Cx. p. quinquefasciatus* are presented in the section on vectors of filariasis.

Culex whitmorei is far less common than the species discussed above. Its life cycle is similar to that of *Cx. tritaeniorhynchus*, although it is most abundant in the middle of the monsoon season from July to September. Larval breeding sites are temporary ground pools and, occasionally, rice fields. The species is mildly endophagic but primarily exophilic. Cattle are the preferred hosts, although man and birds are occasionally attacked.

Culex bitaeniorhynchus is even more uncommon than *Cx. whitmorei* but is widespread throughout the region. The larvae of this species breed in warm, polluted, semi-permanent or permanent pools with abundant algal growth. Partial shading or sunlit areas are preferred. Adults bite early in the evening and are primarily bird feeders, which enhances their role as a zoonotic vector of JE. Seasonal distribution is from the early monsoon season (May or June) to the early dry season (December or January). This species is known to transmit JEV transovarially.

Anopheles subpictus occurs throughout the region. Two sibling species, A and B, have been reported in Sri Lanka and India, with species A predominating in most areas. However, species B is more common in coastal areas, where it may breed in brackish water. Larvae occur in muddy pools near houses, in gutters, in paddy fields, and in borrow pits. *Anopheles subpictus* is primarily zoophilic but feeds readily on man. Its feeding behavior on birds has not been reported. It is exophilic and exophagic but will also feed and rest indoors. This species occurs during the pre-monsoon through post-monsoon seasons (June to November) in northern parts of the region. In southern India and Sri Lanka it occurs year-round. *Anopheles subpictus* feeds indoors primarily from 1800 to 2100 hours and rarely bites indoors after 2300 hours. It can fly up to 2 km from its breeding sites to feed but is not considered a strong flier.

Anopheles hyrcanus is a potential but insignificant JE vector. This is actually a species complex, and the sibling species can only be identified genetically. The complex is widespread throughout the region and can occur at high altitudes. This large, dark mosquito feeds on both humans and animals, but prefers to feed on cattle outdoors. It may feed throughout the night and, occasionally, during the day. It occurs nearly year-round, but densities are generally low. The larvae breed in rice fields, swamps, grassy pools, borrow pits, and ditches. Adults are relatively strong fliers, traveling 3 to 5 km to feed.

Mansonia annulifera is a fairly common species throughout the region. Details on the life cycle and bionomics of this species are presented in the section on filariasis.

Mansonia uniformis is more common than *Ma. annulifera* in many areas, particularly lowlands. However, this species is not as efficient a vector of JEV as *Ma. annulifera*. Details on its life cycle are presented in the section on vectors of filariasis.

Vector Surveillance and Suppression. Most *Culex* vectors of JEV are readily collected in light traps. Animal-baited traps can collect large numbers of zoophilic species. Adults can also be collected with an aspirator from animal sheds and other resting places. JEV can be detected in mosquitoes before outbreaks in humans occur. However, routine surveillance systems based on isolation of virus from mosquito pools are too time- consuming to be of practical use in most military situations. Testing sentinel pigs weekly for seroconversions has been used successfully by public health workers to detect JEV activity in many endemic areas. Implementation of mosquito control is based on the occurrence of seroconversions in pigs, which generally preceed JE outbreaks in the human population by 1 or 2 weeks.

Control of JE vectors over large areas with insecticides is impractical, environmentally unacceptable and prohibitively expensive. Application of insecticides to rice paddies may dramatically reduce larval populations, but they usually recover in within 1 week. Some success in interrupting JEV transmission has been achieved by applying residual insecticides to the interior walls of houses. However, insecticide resistance has become widespread in many vector species (see Appendix C, Pesticide Resistance in South Central Asia). ULV aerosols may be useful in reducing adult populations during periods of epidemic JEV transmission. Long-term control is best achieved by environmental management of breeding sites, such as intermittent irrigation of rice paddies or the use of larvivorous fish. Protection of military personnel is best achieved by use of the **personal protective measures** outlined in TIM 36 and by vaccination.

D. Chikungunya Fever.

Disease produced by the chikungunya virus (*Alphavirus*, family Togaviridae) is characterized by sudden onset, fever, rash, nausea, vomiting, and severe joint pains that may persist as a recurrent arthralgia for months or even years. The incubation period is 3 to 11 days, and the acute illness lasts 3 to 5 days. Minor hemorrhages have been attributed to chikungunya virus disease in India and Southeast Asia. Convalescence is often prolonged. Recovery is usually complete followed by lifelong immunity. Inapparent infections are common, especially in children, among whom clinical disease is rare. Chikungunya can be differentiated from dengue in that pain is predominately located in the joints rather than the muscles, and the febrile period is shorter and usually not diphasic.

Military Impact and Historical Perspective. The disease was first recognized during epidemics that occurred in 1952 among inhabitants of Southern Province, Tanzania. Chikungunya is a Swahili word meaning “that which bends up” and refers to the stooping posture adopted by patients because of the severity of the joint pains. The chikungunya virus was isolated in 1952, but its relationship to other arthropod-borne viruses was not fully determined until the late 1950s. Chikungunya viral infections are a significant military threat because of the lack of vaccines or specific therapy and the abrupt onset of this incapacitating infection. However, the threat appears to be minimal in South Central Asia, due to the limited prevalence of the virus in the region.

Disease Distribution. Chikungunya virus is enzootic throughout tropical Africa, from which it has spread to other parts of the world, primarily Southeast Asia and South Central Asia. India had a history of epidemics from 1824 until 1965, when the virus spread to Sri Lanka. It was estimated that during the 1964 epidemic in Madras nearly 400,000 cases occurred. In a 1973 outbreak in the town of Barsi, Maharashtra State, in central India, the overall morbidity was 37.5% of the residents. This was the last significant outbreak of chikungunya in India. A 1994 study in Calcutta found 12.5% seroprevalence among 51 to 55 year olds but no detectable antibody in children or young adults. These findings suggest that chikungunya virus is disappearing from the human population. Rare outbreaks have occurred in Pakistan, but serological evidence indicates that the virus is currently circulating in there. Chikungunya viral infections have historically been reported from Dinajpur and Lalmonirhat Districts in Bangladesh, but their current status is unclear. The last reported outbreak in Sri Lanka occurred in 1965. Outbreaks are more likely to occur during the rainy season, although incidence in Sri Lanka peaked in June and July, 1 to 2 months after the heavy rains in May. The current endemic status of chikungunya virus in Sri Lanka is unclear.

Transmission Cycle(s). There are no records of clinical disease in domestic animals or wildlife due to infection with chikungunya virus. However, there is a strong body of evidence implicating wild primates as primary reservoir hosts. Antibodies to chikungunya virus have been found in the rhesus monkey,

Macaca mulatta, which is widely distributed in the region. Experimentally infected bonnet macaques, *M. radiata*, develop viremias sufficient to infect *Ae. aegypti*. This macaque is found in peninsular India. The role of these or other macaques in the epidemiology of chikungunya virus in South Central Asia is uncertain. In Asia most human outbreaks of chikungunya have occurred in urban areas, whereas in Africa human infections usually occur in rural areas. The virus can survive for considerable periods in the epidemic human to *Ae. aegypti* cycle, resulting in sporadic outbreaks at irregular intervals.

Vector Ecology Profiles. Most isolations of chikungunya virus in Asia have been made from *Ae. aegypti*. *Aedes albopictus* is an important secondary vector. Most experimentally infected *Culex* spp. and *Anopheles* spp. are refractory to infection with chikungunya virus. Transovarial transmission does not appear to play a role in the maintenance of this virus in nature, since chikungunya fever has disappeared from many urban areas where *Ae. aegypti* has remained abundant. See dengue for the biology of *Ae. aegypti* and *Ae. albopictus*.

Vector Surveillance and Suppression. See dengue.

E. West Nile Fever.

West Nile fever is a mosquito-borne illness characterized by fever, headache, muscular pain, and rash. Serious complications involve the liver and nervous system. The etiological agent, West Nile virus (WNV), is named after the district of Uganda where the virus was first isolated. It is a *Flavivirus* closely related to viruses causing Japanese encephalitis and St. Louis encephalitis. Infection with WNV is most often asymptomatic. The incubation period ranges from 1 to 6 days and clinically resembles a mild dengue-like illness. WNV can be readily isolated from the blood of patients during the first few days of illness. Recovery is complete but may be accompanied by long-term muscle pain and weakness. Permanent sequelae have not been reported. Neurological involvement is most frequent in elderly patients, and most fatal cases have occurred in patients over 50 years old. The mean age of the first 4 fatalities that occurred in New York City during the 1999 outbreak was 81.5 years.

Military Impact and Historical Perspective. WNV was isolated in 1937 and was one of the earliest human arboviral infections to be documented. Undoubtedly, WNV has been the cause of many cases classified as fevers of unknown origin in military personnel. In view of the mild illness and the infrequent occurrence of epidemics, the military impact of this virus is likely to be minor compared with other diseases in South Central Asia. However, a large outbreak in the summer of 1996 in and near Bucharest, Romania, with more than 800 clinical cases and a case-fatality rate approaching 10%, suggests infection with WNV may be more serious than previously thought. Infection with WNV will complicate diagnoses by medical personnel, since West Nile fever cannot be clinically distinguished from many other arboviral fevers.

Disease Distribution. WNV has a broad geographic range and has been isolated in many parts of Africa and Europe. In 1999, it was isolated for the first time in North America. WNV has been reported from a variety of habitats, including coastal, high plateau, forest, and semiarid lands. West Nile fever may be an emerging arboviral disease of public health significance. The epidemiology of WNV in South Central Asia is unclear, but it has been isolated from humans and mosquitoes in India and Pakistan. Studies in the early 1990s indicated that a significant proportion of acute encephalitis cases in Karachi, Pakistan, were caused by WNV infection. Previous serological studies had indicated that WNV was prevalent in Punjab Province. In 1994, antibodies to WNV were found in 33 to 41% of 3 groups of Pakistani military personnel. West Nile fever occurs during the period of maximum activity of mosquito vectors, usually during periods of high temperature and rainfall. In endemic areas of Pakistan, the May to July period has the maximum potential for WNV transmission. Environmental factors, including human activities that enhance vector populations (irrigation, heavy rains followed by flooding, higher than normal temperatures), increase the potential for an outbreak of this and other mosquito-borne diseases.

Transmission Cycle(s). WNV has been isolated from numerous wild birds and, occasionally, from mammals. Serological surveys have demonstrated WNV antibodies in wild and domestic bird species, wild mammals such as lemurs, rodents and bats, and domestic animals such as camels, horses, mules, donkeys,

goats, cattle, water buffalo, sheep, pigs and dogs. Infected pigs develop low titers of WNV, so they do not act as amplifying hosts for this virus, as they do for Japanese encephalitis virus. WNV has been isolated from bats in India. Equines can become seriously ill and die from infection with WNV.

Birds are considered to be the primary reservoir for WNV and may reintroduce the virus during seasonal migrations. However, the absence of migrational routes between southeastern India and the African-Middle East region suggests an isolated antigenic group of WNV. In Pakistan, 19 of 31 species of birds captured in a forest plantation were found to have WNV antibody. The situation is less clear in India because of the presence of JEV antibodies. Birds usually do not show any symptoms when infected with WNV, though illness and death have been observed in pigeons and crows. Infections in most mammals fail to produce viremias high enough to infect potential vectors.

WNV has been isolated from many species of mosquitoes in nature, especially *Culex* spp., which are recognized as the primary vectors. WNV has also been recovered from bird-feeding ticks and mites, and experimental transmission of WNV has been observed in *Ornithodoros* spp. and several species of hard ticks. A natural bird-tick zoonotic cycle has been suggested, but the role of ticks in the natural transmission of WNV has not been well defined. Mosquitoes are clearly implicated in the transmission of WNV to humans. WNV replicates quickly in mosquitoes when temperatures exceed 25^{o}C. Infected mosquitoes can transmit WNV for life. Experimental transmission of WNV by *Aedes albopictus* has been reported. Field and experimental evidence suggests that transovarial transmission may occur.

Vector Ecology Profiles. Primary vectors in South Asia are *Culex pipiens quinquefasciatus* (*Cx. pipiens fatigans*), *Cx. tritaeniorhynchus*, and *Cx. vishnui*. Additonal vectors include *Cx. pseudovishnui* and possibly *Anopheles subpictus*. Of the primary vector species, *Cx. p. quinquefasciatus* is considered to be the best vector of West Nile fever.
Culex p. quinquefasciatus is the most common species throughout the region that feeds readily on both man and birds, which is essential to linking the zoonotic and epidemic cycles of the virus. Details on the bionomics of this species are presented in the section on filariasis.

Culex tritaeniorhynchus breeds primarily in the pre-monsoon and monsoon periods (mid-May through mid-September) in northern parts of the region. In southern parts of the region, it may breed year-round. Details on the bionomics of this species are presented in the section on Japanese encephalitis. A low rate of experimental transovarial transmission of WNV has been reported for this vector.

Culex vishnui and *Cx. pseudovishnui* are abundant during the pre-monsoon and monsoon seasons. Details on the bionomics of these species are presented in the section on Japanese encephalitis.

Anopheles subpictus occurs widely through most of the region. Two sibling species, A and B, have been reported in Sri Lanka and India, with species A predominating in most areas. However, species B predominates in coastal areas. *Anopheles subpictus* is primarily zoophilic but also feeds aggressively on man. Feeding behavior on birds has not been reported, but birds are the reservoir of WNV, and West Nile viral isolates have been obtained from this mosquito. Details on the distribution and bionomics of this species are presented in the section on malaria. A list of South Central Asian mosquitoes and their distribution can be found in Appendix A.1.

Vector Surveillance and Suppression. Epidemics of West Nile fever are infrequent, and public health officials in South Central Asian countries can rarely justify continued long-term surveillance for virus activity when considering other health care demands. Pigs and chickens have been used as sentinel hosts to detect WNV activity. Surveillance for dead crows may also be a useful indicator of WNV activity in an area. Reduction of mosquito populations by ULV spraying may be a means of disease control during outbreaks. The most feasible long-term control strategies involve reducing vector breeding by environmental management techniques, especially in agricultural areas under extensive irrigation. Experimental evidence suggests that vaccination with Japanese encephalitis vaccine may provide protection against WNV. **Personal protective measures** to prevent mosquito bites are the most practical means of avoiding infection with WNV. Consult TIM 13, Ultra Low Volume Dispersal of Insecticides by Ground

Equipment; TIM 24, Contingency Pest Management Pocket Guide; and TIM 40, Methods for Trapping and Sampling Small Mammals for Virologic Testing. Also see vector surveillance and suppression for malaria.

F. Sindbis Virus.

Sindbis virus belongs to the genus *Alphavirus*, family Togaviridae. It is closely related to the Western equine encephalitis complex. The incubation period is less than a week and symptoms may include fever, headache, rash, and joint pain. Syndromes resulting from Sindbis virus infection have been called Ockelbo disease in Sweden, Pogsta disease in Finland, and Karelian fever in the former Soviet Union. No fatal cases have been reported.

Military Impact and Historical Perspective. Sindbis virus was first isolated in 1952 from *Culex* mosquitoes collected in the village of Sindbis north of Cairo. It was subsequently reported from Europe, Asia, sub-Saharan Africa and Australia. A role in human disease was recognized in 1961, when Sindbis virus was isolated from patients with fever in Uganda. Human epidemics due to Sindbis virus were documented in South Africa in the late 1960s, and clusters of cases with fever, arthralgia and rash have been observed in Sweden. Although outbreaks of Sindbis virus in areas of northern Europe have caused significant human morbidity, this disease is expected to have a minor impact on military operations in South Central Asia. Clinically, human infections could be confused with illness produced by dengue, chikungunya and West Nile viruses.

Disease Distribution. Sindbis virus is one of the most widely distributed of all known arboviruses. Studies have demonstrated Sindbis virus transmission in most areas of the Eastern Hemisphere. Very little is known about the epidemiology of Sindbis virus in South Central Asia and its role in human disease. The virus has been isolated from birds, *Culex* mosquitoes and a dermanyssid mite in India, and antibodies to Sindbis virus have been found during surveys of birds in Pakistan.

Transmission Cycle(s). A wide range of wild and domestic vertebrate species are susceptible to infection with Sindbis virus. Most experimentally infected wild bird species easily produce viremias high enough to infect several different species of mosquitoes. Wild and domestic birds are considered the main enzootic reservoir. Although several species of domestic mammals can become infected with Sindbis virus, there is no evidence that these infections result in significant illness. Evidence implicates bird-feeding mosquitoes of the genus *Culex* as the vectors of Sindbis virus in enzootic and human infections. However, viral isolations and transmission experiments have shown that some *Aedes* spp., which are less host specific and feed readily on both birds and humans, may be important as vectors linking the enzootic cycle and human infection. Mechanisms that allow the virus to overwinter and survive between periods of enzootic transmission in temperate climates have not been identified. Outbreaks of Sindbis fever epidemics in northern Europe are believed to be caused by the introduction of the virus from Africa by migratory birds.

Vector Ecology Profiles. *Culex bitaeniorhynchus* and *Cx. vishnui* have been implicated as vectors of Sindbis virus in South Central Asia. See Japanese encephalitis for the biology of these species. A complete list of mosquitoes and their distribution in South Central Asia appears in Appendix A.1.

Vector Surveillance and Suppression. See West Nile fever and malaria.

G. Sand Fly Fever.

The sand fly fever (aka: Papatasi fever; Three-day fever) group of viruses (*Phlebovirus*, Bunyaviridae) contains at least 7 immunologically related types. Naples, Sicilian and Toscana are normally associated with sand fly fever. The virus produces an acute febrile illness lasting 2 to 4 days and is commonly accompanied by headache and muscle pain. There is usually no mortality, nor are significant complications associated with infections of Naples or Sicilian viruses, although weakness and depression may persist a week or more after acute illness. However, Toscana virus may cause central nervous system disease in addition to fever. Most infections are acquired during childhood in endemic areas. The clinical disease in children is generally mild and results in lifelong immunity to homologous strains.

Military Impact and Historical Perspective. Sand fly fever has been an important cause of febrile disease during military operations since at least the Napoleonic Wars. In 1909, an Austrian military commission first reported that an infectious agent found in the blood of infected soldiers caused this fever, and that the vector was the sand fly. During World War II, sand fly fever attained importance in Allied and Axis forces in the Mediterranean theater by incapacitating large numbers of men for periods of 7 to 14 days. The disease was first recognized in U.S. forces in North Africa in April 1943. Although several thousand cases were reported from 1943 to 1945 in the Mediterranean theater, the incidence of sand fly fever was undoubtedly underestimated. There were nearly 3,000 cases of sand fly fever recorded in U.S. Army personnel from 1942 to 1945 in the China-Burma-India theater. Most cases occurred in India. In 1943, several explosive outbreaks were confined to small areas. Up to 40% of the members of one command were infected at one time. Many thousands of cases of sand fly fever were probably recorded as fevers of unknown origin, since most medical officers were unfamiliar with the disease and specific tests for diagnosis were not available. In sharp contrast to World War II, there were no reports of sand fly fever among coalition forces during the Persian Gulf War. The military significance of sand fly fever is magnified because of its short incubation period, which may result in large numbers of nonimmune troops being rendered ineffective early in an operation, while endemic forces would be largely immune and unaffected.

Disease Distribution. Sand fly fever is focally distributed countrywide in Afghanistan, Bangladesh, India and Pakistan. Both the Naples and Sicilian viruses are circulating in the region. Viral isolates were made from febrile Soviet troops operating in Paravan Province, Afghanistan, in 1987. Transmission may occur year-round in tropical and subtropical areas, but is limited to the warmer months of the year in cooler northern areas. Risk of transmission is elevated in village and periurban areas. The widespread application of insecticides inside houses during the intense malaria control programs of the 1960s and 1970s also reduced the abundance of sand flies and the incidence of sand fly-borne disease. However, the relaxation of malaria control efforts in recent years has increased the potential for transmission of sand fly fever. In the late 1980s, 27 to 70% of 3 groups of Pakistani military personnel admitted to the hospital during outbreaks of hepatitis or febrile illness had antibodies to sand fly fever viruses.

Transmission Cycle(s). No vertebrate reservoir has been clearly established, but small rodents, especially gerbils, may serve as reservoirs. Infected humans can infect sand flies and thus have an amplifying effect during epidemics. Humans with high levels of virus in the blood more easily infect sand flies. The principal reservoir mechanism appears to be transovarial transmission. The virus is most efficiently replicated in the sand fly vector and transmitted when temperatures exceed 25 ^{o}C. Infected sand flies remain infective for life and are not harmed by the virus.

Vector Ecology Profiles. The primary vector species are *Phlebotomus papatasi* and *P. argentipes*. These species are widely distributed in most countries in the region except the Maldives. Both species are probably complexes, with differing sibling species in Southeast Asia and South Central Asia. Possible vectors include *Sergentomyia* spp., such as *S. babu*, although viral isolations are rarely reported from this genus in South Central Asia.

Phlebotomus papatasi is a highly anthropophilic species that is widely distributed throughout the region, particularly in lowlands. *Phlebotomus papatasi* is not found above 1,800 m in Afghanistan, suggesting that its distribution is limited in that country, as well as in Nepal and Bhutan. In northern parts of its range, populations of this species and of *P. argentipes* peak in June, during the early part of the monsoon season.

Some *P. papatasi* are autogenous; that is, they are capable of producing small numbers of eggs without a bloodmeal, at least during the first gonotrophic cycle. After a bloodmeal, females deposit eggs, 30 to 70 at a time, in rodent burrows, poultry houses, masonry cracks, rock crevices, leaf litter or moist tree holes. Eggs hatch in 1 to 2 weeks, and larvae develop in warm, moist microhabitats that provide abundant organic matter for food. In military fortifications, larvae may live in the cracks between stacked sandbags. There are 4 larval instars, and 4 to 8 weeks are required to reach the pupal stage. Fourth-instar larvae may diapause for weeks or months if environmental conditions are excessively cold or dry. Alternatively, if environmental conditions improve, diapause may be quickly broken. Pupation occurs in the larval habitat.

There is no cocoon; rather, the pupa is loosely attached to the substrate by the cast skin of the 4th larval instar.

Phlebotomus papatasi feeds most intensely at dusk and dawn, with some feeding continuing sporadically through the rest of the night. *Phlebotomus papatasi* and *P. argentipes* tend to be exophilic through the southern part of their range (i.e., India, Pakistan and Bangladesh) but endophilic in Nepal and Afghanistan. They also are more zoophilic in the southern part of their range, preferring cattle to people. Zoophilic and exophilic populations of these species may have been selected for by the intensive malaria spraying that has occurred in Bangladesh, India and Sri Lanka. Nevertheless, both species readily attack man throughout the region.

Phlebotomus papatasi and *P. argentipes* follow odor plumes to their hosts. Like mosquitoes only female sand flies suck blood, but both sexes feed on plant sugars and nectar. Besides humans, female sand flies feed on the blood of a variety of birds and mammals, commonly cattle, dogs, mice, voles, gerbils and desert hedgehogs. The preference of *P. argentipes* for cattle has been well documented; however, in some areas populations of this species are highly anthropophilic. This and other observations on the biology of *P. argentipes* suggest that it is a species complex.

On humans, sand flies feed on exposed skin around the head, neck, legs, and arms. Female sand flies will crawl under the edges of clothing to bite skin where repellent hasn't been applied. Persons newly exposed to the bites of sand flies often experience a severe urticarial reaction until they become desensitized. Sand flies feed outdoors or indoors and readily penetrate ordinary household screening. After engorgement, *P. papatasi* and other sand flies rest briefly on objects near their host, then move to gerbil burrows or other cool, moist environments to lay eggs. They also rest in caves and other areas that are relatively cool and shaded during the daytime.

Most species of sand flies are weak fliers and do not travel in wind that exceeds a few kilometers per hour. *Phlebotomus papatasi* may be active at a low relative humidity of 45 to 60%, but other vector species require 75 to 80% relative humidity in order to fly and feed. Sand flies normally fly in short hops, which usually limits their feeding radius to 100 to 200 m from pupation sites. However, unengorged females may occasionally disperse as far as 1.5 km. Mating dances occur on the ground or on animal hosts such as cows, usually at dusk, with males landing first, followed by females. A list of sand fly species and their distribution in South Central Asia appears in Appendix A.2.

Vector Surveillance and Suppression. Because sand flies are small and retiring, specialized methods are required to collect them. The simplest method is active searching of daytime resting sites with an aspirator and flashlight, but this method is very labor intensive. Human-landing collections are an important method of determining which species are anthropophilic. Sticky traps (paper coated with a sticky substance or impregnated with an oil such as castor oil, mineral oil or olive oil) are used to randomly capture sand flies moving to or from resting places. Traps can also be placed at the entrances of animal burrows, caves and crevices, in building debris, and in local vegetation where sand flies are likely to rest during daytime hours. A variety of light traps have been used to collect phlebotomines, but their effectiveness varies according to the species being studied and the habitat. Light traps are inefficient in open desert. Light traps used for mosquito collection should be modified with fine mesh netting in order to collect sand flies. Traps using animals as bait have been also devised. Collection of larvae is extremely labor intensive and usually unsuccessful because specific breeding sites are unknown or hard to find and because females deposit eggs singly over a wide area. Emergence traps are useful for locating breeding sites. Identification of sand flies requires a microscope and some training; however, with a little experience, sorting and identification by color and size will suffice using minimal magnification. For accurate species identification, laboratory microscopes with 100x magnification are required.

Because of their flight and resting behavior, sand flies that feed indoors are very susceptible to control by residual insecticides. When sand flies are exophilic or bite away from human habitations, control with insecticides is impractical, although the application of residual insecticides to a distance of 100 m around encampment sites may be helpful. Some success in reducing vector populations has been achieved by controlling the rodent reservoir or host population. Selection of encampment sites without vegetation or

rock outcroppings that harbor rodents is important. Cleanup and removal of garbage and debris that encourage rodent infestation are necessary for longer periods of occupation. Pets must be strictly prohibited, because any small desert rodent and/or local dog may be infected with leishmaniasis or other diseases.

Sand flies are able to penetrate standard mesh screening used on houses and standard mesh bednets (seven threads per cm or 49 threads per sq cm). These items should be treated with permethrin to prevent entry. Fine mesh (14 threads per cm or 196 threads per sq cm) bednets can be used to exclude sand flies, but these are uncomfortable under hot, humid conditions because they restrict air circulation. The use of repellents on exposed skin and clothing is the most effective means of individual protection. Insect repellent should be applied to exposed skin and to skin at least 2 inches under the edges of the BDU to prevent sand flies from crawling under the fabric and biting. The use of **personal protective measures** (see TIM 36) is the best means of preventing sand fly-borne disease.

H. Crimean-Congo Hemorrhagic Fever.

Crimean-Congo Hemorrhagic Fever (CCHF) is a zoonotic disease caused by a tick-borne virus of the genus *Nairovirus*, family Bunyaviridae. The disease is characterized by febrile illness with headache, muscle pain and rash, frequently followed by a hemorrhagic state with hepatitis. The mortality rate can exceed 30%. The incubation period ranges from 3 to 10 days. CCHF may be clinically confused with other hemorrhagic infectious diseases. Humans are the only natural host of CCHF virus in which disease has been confirmed. The ratio of apparent to inapparent infections suggests that 1 of every 5 persons infected develops clinical illness.

Military Impact and Historical Perspective. Descriptions of a disease compatible with CCHF can be traced back to antiquity in eastern Europe and Asia. CCHF was first described in soldiers and peasants bitten by ticks of the genus *Hyalomma* while working and sleeping outdoors on the Crimean peninsula in 1944. The virus was first isolated in 1967. Since there are no available treatment regimens of proven value and recovery from CCHF can be protracted, military personnel with CCHF would require significant medical resources. However, there is limited evidence that ribavirin may be useful in the treatment of CCHF infections.

Disease Distribution. CCHF virus has the widest geographic distribution of any tick-borne virus. It is enzootic in the steppe, savanna, semi-desert and foothill environments of eastern and central Europe, Russia, parts of Asia, and throughout Africa. These are favored habitats of xerophilous *Hyalomma* ticks. Several Eurasian CCHF epidemics have taken a great toll of human life. In recent years, cases of CCHF have tended to be sporadic, with most reported from Bulgaria and South Africa. Bulgaria recorded 1,410 cases, primarily in agricultural workers, between 1953 and 1992. However, CCHF is underdiagnosed in many countries due to lack of appropriate medical and laboratory services.

CCHF virus is likely enzootic in widely distributed discrete foci throughout South Central Asia. Vector ticks are common on domestic animals throughout the region. Although no human cases have been reported from India and Nepal, CCHF viral antibodies have been found in domestic animals and humans. Human cases have been reported from the Muree Hills near Islamabad, Changa Manga National Forest in Punjab Province, and from Baluchistan Province in Pakistan. In February 1998, 4 cases occurred in the same family of sheep herders in a village in the Kohlu area of Baluchistan Province. An outbreak of CCHF occurred in a village in the district of Rustaq, Province of Takar, Afghanistan, in March 1998. A total of 19 cases, including 12 fatalities, were reported. Another suspected outbreak of CCHF involving 27 cases occurred in June 2000 in an isolated village in Gulan District, Herat Province, Afghanistan.

Transmission Cycle(s). CCHF virus has been isolated from at least 30 species of ticks. From experimental evidence, it appears that many tick species are capable of transmitting the virus, but members of the genus *Hyalomma* are the most efficient vectors. The aggressive host-seeking behavior of adult hyalommine ticks makes them ideal vectors. The highest prevalence of antibodies in wild and domestic reservoirs has been found in areas where *Hyalomma* ticks are common. Domestic ruminants generally acquire infection early in life. Viremia in livestock is short-lived and of low intensity. Antibodies or virus

have also been found in a variety of small mammals, including hedgehogs, hares, bats and rodents. Most birds are resistant to infection with CCHF virus. In Pakistan, CCHF viral antibodies have been found in *Rattus rattus*, *R. norvegicus*, the jird *Meriones libycus*, and the naked soled-gerbil *Tatera indica*. Infected wild and domestic animals show no serious signs of disease. Transovarial transmission of virus in vector ticks is an important reservoir mechanism. The wide range of tick species from which CCHF virus has been isolated, the diversity of life cycles and habitats of these ticks, and the uncertain involvement of various vertebrates have contributed to a poor understanding of the complex transmission and maintenance cycles of CCHF. Historically, the recognition of CCHF virus enzootic foci has been characterized by an unpredictable and sudden occurrence of human CCHF cases in presumably nonenzootic areas.

Persons occupationally exposed to domestic animals, such as animal husbandry and abattoir workers, are at greatest risk of infection. Humans acquire CCHF virus from tick bites, from contamination of broken skin or mucous membranes with crushed tissues or feces of infected ticks, or from contact with blood or other tissues of infected animals. CCHF virus is highly infectious, and nosocomial infection of medical workers has been important in many outbreaks. In December 1994, 3 medical workers in a private hospital in Quetta, Pakistan, contracted CCHF after surgery on a bleeding patient who later died.

CCHF virus loses infectivity shortly after the death of an infected host. There is no indication that consumption of meat processed according to normal health regulations constitutes a hazard.

Vector Ecology Profiles. CCHF virus has been isolated from *Hyalomma marginatum marginatum* in Afghanistan and from *Hyalomma anatolicum anatolicum* and *Boophilus microplus* in Pakistan.

The principal vector is *Hyalomma a. anatolicum*, which is a widely distributed tick species in Pakistan and India. It inhabits steppe, semi-desert, and savanna biotopes.
Adult *H. a. anatolicum* prefer to feed on cattle, horses, sheep, goats, dogs and, occasionally humans. Female ticks generally oviposit after leaving the host, laying thousands of eggs on open ground or on vegetation. Females die soon after oviposition is complete. Adults of this vector wait in rodent burrows or on plants and quickly move toward hosts as they appear. Adult females may remain on the host and feed for 4 to 6 days. Immature ticks generally climb vegetation or other objects in order to quest for hosts. Nymphs remain on the host for several days.

Hyalomma a. anatolicum is among the world's hardiest ticks and can easily survive extremes of heat, cold, and aridity for a year or more. Under ideal conditions the life cycle can be completed in a year. Over the centuries, this tick has been widely distributed along routes of trading caravans and cattle drives. *Hyalomma a. anatolicum* is frequently a one-host tick and remains on the same animal throughout its life, an unusual habit in hyalommine ticks. This species is important in human outbreaks of CCHF because of its broad host range, wide distribution, and aggressive host-seeking behavior. In addition, the virus is passed both transstadially and transovarially, which makes this species an important reservoir as well as a vector. Cracks in stone or clay stable walls, courtyards, and feedlots often harbor these ticks. Nymphs tend to feed on the ears of their hosts. In tropical parts of South Central Asia, tick development may continue year-round.

Hyalomma marginatum is another possible vector, based on its wide distribution in the region. *Hyalomma marginatum* inhabits woods and meadows, primarily in rolling terrain with cattle. This two-host tick also parasitizes horses. *Hyalomma marginatum* begins to appear on cattle in February and becomes progressively more abundant with each succeeding month through May, but populations drop sharply during the monsoon season. This species is one of the hardiest, most cold-resistant ticks in the world. It also can survive arid conditions quite successfully. Immature stages quest actively from rodent burrows or grasses and feed primarily on small mammals (rodents, hedgehogs, and hares) and birds (usually rooks and magpies). Nymphs feed from 5 to 8 days before dropping off their hosts, then molt and await their adult host as they quest from tall grasses, often in pastures. In addition to cattle and horses, adults feed on goats, dogs, sheep and, occasionally, humans. After feeding for 6 to 12 days, females drop off their hosts and oviposit thousands of eggs. Under ideal conditions, the life cycle can be completed in a single year, although adults often overwinter and begin laying eggs in the spring. Under adverse conditions, the life

cycle can be extended for 2 or more years. Transovarial and transstadial transmission of CCHF occur efficiently in this species, making it an important reservoir as well as vector of CCHF.

Boophilus microplus is a possible secondary vector, but it is not as important as hyalommines in transmitting CCHF. This species is widely distributed in virtually every country in South Central Asia. *Boophilus microplus* is a one-host tick that is primarily a cattle feeder, but it also feeds on other herded ungulates and, less frequently, on man. After feeding and mating, females may rest up to a month before laying 2,000 to 3,000 eggs. These hatch in about 2 weeks if relative humidity is 70% or more. Larvae attach to a host in the spring and feed for 2 to 4 days, then molt to the nymphal stage while still on the host. The nymph feeds on the host for about a week, after which it exhibits a short quiescent period and molts to the adult stage. The life cycle generally occurs in 1 season, sometimes in 6 weeks or less when temperatures and humidity are high.

Vector Surveillance and Suppression. Military personnel should conscientiously use **personal protective measures** to prevent tick bites (see TIM 36). Frequent self-examination and removal of ticks are important. Ticks should be handled as little as possible and not crushed. Troops should not sleep, rest or work near rodent burrows, huts, abandoned rural homes, and livestock or livestock enclosures. Close contact with livestock and exposure to locally butchered animals should be avoided. Serological surveys of domestic animals are the most practical and economical surveillance systems for CCHF virus. Sheep, goats and cattle have exhibited high antibody prevalence to the virus in areas where human disease has been documented.

An inactivated mouse-brain vaccine against CCHF has been used in eastern Europe and the former Soviet Union on a small scale. The FDA has not approved a vaccine for human use. A purified modern vaccine will probably not be developed in view of the limited potential demand.

I. Boutonneuse Fever.

This tick-borne typhus, (aka: Mediterranean tick fever; Mediterranean spotted fever; Marseilles fever; African tick typhus; Kenya tick typhus; India tick typhu)s is a mild to severe illness lasting a few days to 2 weeks. Clinical symptoms begin 6 to 10 days after the bite of an infected tick and include fever, headache and muscle pain. Boutonneuse fever is caused by *Rickettsia conorii* and closely related rickettsial organisms. Different strains of *R. conorii* isolated from ticks and humans indicate that this pathogen has substantial genetic and antigenic diversity. The common name of this disease comes from the button-like lesions, 2 to 5 mm in diameter, that develop at tick attachment sites. With antibiotic treatment, fever lasts no more than 2 days. The case fatality rate is usually very low, even without treatment.

Military Impact and Historical Perspective. Historically, boutonneuse fever has not significantly interfered with military operations. Sporadic cases among combat troops can be expected in limited geographic areas. The severity of illness will be dependent upon the strain of *R. conorii* contracted. Because the spotted fevers are regional diseases involving different species or strains of *Rickettsia*, military medical personnel newly assigned to an area may be unfamiliar with them, and diagnosis may be delayed.

Disease Distribution. Boutonneuse fever is widespread in countries bordering the Mediterranean, and most countries of Africa. Along the European Mediterranean coast, the seroprevalence of boutonneuse fever in humans varies from 4.2 to 45.3%, depending on the geographical region. Expansion of the European endemic zone to the north is occurring because North European tourists vacation along the Mediterranean with their dogs, which acquire infected ticks that are then brought home. *Rickettsia conorii* has been isolated from India, Nepal, Pakistan and Sri Lanka, where it is known as Indian tick typhus. *Rickettsia sibirica* circulates in Afghanistan, Nepal and Pakistan, and the clinical disease it causes is called North Asian or Siberian tick typhus. Only sporadic human cases are reported, although the seroprevalence to *R. conorii* was 10.9 % in the general population in northern Pakistan (date unspecified). The highest rate was in laboratory animal handlers (31.2%), followed by vets (21.2%), and farmers (14.8%). The incidence of *R. conorii* was highest in Mangla Township (33.3%), followed by Peshawar (25.6%), Rawalpindi/Islamabad (17.9%), Mansehra (13.4%), and Abbottabad (12%). Incidence of *R. cornorii* appears to be higher in southern India than in other parts of the country. A Sri Lankan study in the early

1990s found *R. conorii* antibodies in 20% of the goat sera and 30% of the cattle sera that were tested. The areas of highest risk of transmission of boutonneuse fever in South Central Asia are depicted in Figure 6.

Figure 6. Distribution of Boutonneuse Fever in South Central Asia.

FIG. 6. DISTRIBUTION OF BOUTONNEUSE FEVER IN SOUTH CENTRAL ASIA (DARK SHADING).

Transmission cycle(s). The disease is maintained in nature by transovarial passage of rickettsiae in ticks, primarily the brown dog tick, *R. sanguineus*, although almost any ixodid tick may harbor the pathogen. Enzootic infection in dogs, rodents, hares and other animals is usually subclinical. Dogs do not sustain infection for long but are significant because they bring ticks into close contact with people. Transmission to humans is by the bite of infected ticks. Contamination of breaks in the skin, mucous membranes, or eyes with crushed tissues or feces of infected ticks can also lead to infection. Close association with domestic dogs in endemic areas is a risk factor for boutonneuse fever.

Vector Ecology Profiles. *Rhipicephalus sanguineus* is the principal vector in South Central Asia. Although other vectors are possible, no substantial body of evidence has incriminated other ticks as significant vectors to humans in the region. During a 1980 study in the Shimoga district of Karnataka,

India, 35 isolates of *R. conorii* were recovered from 3 dominant tick species, *Haemaphysalis spinigera*, *H. papuana kinneari*, and *H. turturis*. *Rickettsia conorii* antibodies were found in several species of small and large mammals but were absent from 488 human sera tested in the area. *Rickettsia conorii* was isolated from *Boophilus microplus* and *Rhipicephalus haemaphysaloides* in the Pune District of Maharashtra, India. *Rickettsia sibirica* has been isolated from *Hyalomma marginatum*, *H. detritum* and *R. sanguineus* in western Pakistan. Such a wide range of vectors may result from the existence of previously unrecognized species or serotypes that have been confused with *R. conorii*.

Rhipicephalus sanguineus is reported throughout the entire region. Vector ticks, and hence the disease, tend to be more urban than rural in distribution, because the principal vectors are associated with canine hosts found most abundantly in urban areas.
Rhipicephalus sanguineus, the brown dog tick, feeds primarily on dogs but also on camels, gerbils and, occasionally, humans. It is a three-host tick, with larval and nymphal stages preferring to feed on rats or dogs, while adults feed primarily on dogs, and opportunistically on humans. Larvae and nymphs of *R. sanguineus* spend 3 to 6 days feeding on a host, then drop off to molt. Males feed on their hosts for 3 to 5 days but do not produce sperm until after engorgement. After mating on the host animal, the female feeds for 7 to 15 days, then drops off the host to lay eggs. Females lay 100's of eggs, generally in the dens of host animals, usually canines, or in the cracks and crevices of infested houses. Eggs may require 10 to 20 days to hatch. Adult *R. sanguineus* are passive in their host-questing activity, rarely moving more than 2 m to find a host. This species requires a humid microhabitat, which it can often find in the dens of its hosts, even in the generally arid climates in parts of India, Pakistan and Afghanistan. Appendix A.3. lists the known tick species and their reported distribution in South Central Asia.

Vector Surveillance and Suppression. Personal protective measures (see TIM 36) afford the best protection against boutonneuse fever. In endemic areas domestic dogs are commonly infested with the brown dog tick. Troops should not be allowed to feed, befriend or adopt local dogs as pets.

J. Kyasanur Forest Disease.

The etiological agent, Kyasanur Forest disease (KFD) virus, is closely related to the virus causing tick-borne encephalitis and Omsk hemorrhagic fever and is a member of the tick-borne flavivirus complex. The incubation period of KFD is 2 to 7 days. Onset is sudden, with chills, frontal headache and high fever. The severe muscle pain that follows is reminiscent of dengue. Diarrhea and vomiting occur by the third or fourth day. Severe cases are associated with hemorrhages but no rash. Bleeding occurs from the gums, nose, gastrointestinal tract, uterus and lungs. The case fatality rate is 5 to10%. Hemorrhage begins as early as the third day, but the majority of cases run a full course without hemorrhagic symptoms. The convalescent phase of the disease is prolonged and associated with extreme muscle weakness. Patients need 1 month or more to recover. Humans circulate the virus for at least 6 days in their blood; consequently, virus isolation is easily accomplished.

Military Impact and Historical Perspective. In March 1957, several deaths were noted among langur and macaque monkeys in forest areas of the Shimoga District of Karnataka (then Mysore) State, southern India. Not only were monkeys dying in large numbers, but cases of febrile illness were observed among humans in the area. The zoonosis was first suspected to be sylvan yellow fever, but studies by the Virus Research Center, Poona, India, led to the isolation of several strains of virus from humans, monkeys and ticks. The etiologic agent was named Kyasanur Forest disease virus after the locality from which it was first isolated. Clinical cases have so far been limited to foci within Karnataka State. KFD is unlikely to have significant military impact due to its limited distribution. Differential diagnosis may be confusing to military personnel unfamiliar with the disease. Many laboratory workers have been infected with KFD virus, probably through inhalation of virus in aerosols. The possibility of transmission of KFD from patients to medical staff must be considered when treating patients.

Disease Distribution. Since the first record of the disease in 1957, epidemics of KFD have occurred nearly every year. The number of cases average about 400 to 500 per year. In 1983 and 1984, over 2,000 cases with 250 deaths occurred when a patch of virgin forest was being cleared in South Kanara District. A large number of lumbermen contracted the disease, and a number of villagers also fell ill. Ultimately, the

clearing of the forest had to be suspended by the authorities. After KFD virus was first discovered in 1957, the known range of the virus was limited to within 100 sq km of Sagar and Sorab Taluks in Shimoga District. During subsequent years, KFD spread rapidly to a number of other foci in the Chikmagalur, Dakshina, Kannada, North Kanara, Shimoga and South Kanara Districts. However, clinically and virologically proven cases have been limited to Karnataka State (Figure 7). Serological surveys have found KFD viral antibodies in humans, equines, camels, goats and cattle in some areas of Gujarat State on the east coast near the border with Pakistan. No human disease has been reported from these areas, but it is intriguing that this arid to semiarid area, geograpically removed from Karnataka, may have KFD virus or a closely related virus.

Figure 7. Distribution of Kyasanur Forest Disease in South Central Asia.

FIG. 7. DISTRIBUTION OF KYASANUR FOREST DISEASE IN SOUTH CENTRAL ASIA (DARK SHADING)

KFD exhibits a clear seasonal distribution. The majority of the people in KFD areas are engaged in agriculture from May to December. The monsoon or rainy season is from June to September. During the dry season (November to May), villagers collect firewood and other forest products and take cattle to the forest for grazing. Almost all human cases have a history of forest exposure. The epidemic period begins

in November or December, peaks from January to April, and declines by May. Monkey epizootics show a similar pattern, and nymphal stages of vector ticks are most abundant in the forest during this period. It is believed that *Ixodes* species and small mammals maintain the virus in nature during the monsoon season. Extensive deforestation has occurred in Karnataka State since the discovery of KFD.

Transmission Cycle(s). Monkeys are the principal amplifying hosts. KFD virus has been isolated from the black-faced langur monkey, *Presbytis entellus*, and the South Indian macaque, *Macaca radiata*. The monkeys circulate the virus in very high titers for about 7 days. The langur usually dies from the infection, although the macque often survives. In the KFD area, 36 species of ticks have been recorded. Of the 15 *Haemaphysalis* present in the area, virus has been isolated from 10. Over 90% of viral isolations have come from *H. spinigera*, and this is the predominant tick on the forest floor. Monkeys in the KFD area are not entirely arboreal. They spend enough time on the ground to become infested with ticks. Epizootics in monkeys and epidemics correspond with the nymphal season of *Haemaphysalis* ticks. Several species of small mammals circulate the virus in sufficiently high titers to infect ticks, especially Blanford's rat (*Rattus blanfordi*), the jungle striped squirrel (*Funambulus tristriatus tristriatus*), the giant flying squirrel (*Petaurista petaurista philippensis*), the Indian crested porcupine (*Hystrix indica*), and the common house shrew (*Suncus murinus*). Birds do not appear to play a significant role in the epidemiology of KFD. Cattle and goats are the most numerous domestic animals in KFD areas but don't circulate the virus in titers high enough to infect ticks, and no disease manifestations have been recorded in other domestic animals such as sheep, dogs or cats. Cattle are important in maintaining high populations of *Haemaphysalis* ticks. KFD virus isolations have been made from bats and the soft ticks (*Ornithodoros chiropterophila*) infesting them. However, the ixodid vectors of KFD are rarely found on bats, and it appears unlikely that bats are a reservoir of infection to man.

Vector Ecology Profiles. The predominant vector species is *Haemaphysalis spinigera*, which is widely distributed throughout southern India and Sri Lanka. This species is the source of about 95 % of the Kyasanur viral isolates. Other species from which the virus has been isolated are, in order of frequency of isolations, *H. turturis, H. kinneari, H. kyasanurensis, H. wellingtoni, H. bispinosa, H. minuta, H. cuspidata, H. intermedia* and *H. aculeata*. The virus has also been isolated from *Ixodes ceylonensis, I. petauristae, Dermacentor auratus* and *Rhipicephalus haemaphysaloides*. Of the above species, only *H. spinigera, H. turturis, I. petauristae* and *I. ceylonensis* are thought to be significant vectors. The other species, some of which may be occasional vectors, will not be discussed.

Haemaphysalis spinigera is a three-host tick. The species is widely distributed in tropical forests in the region. The larval tick feeds on birds or small mammals such as shrews, rats, or porcupines. Attachment during feeding lasts from 2 to 6 days. Nymphs feed from 2 to 6 days on these same hosts, but also feed on monkeys and humans. Adults ticks feed for 6 to16 days on monkeys, humans, and cattle.

Oviposition occurs in the leaf litter on the forest floor, with females depositing all their eggs within 5 to11 days. The most favorable period for egg survival is from October to December - after the monsoon, but before the onset of cold, dry weather. Hatching occurs about 10 days after oviposition. Larvae quest on small plants or stems and leaf tips. Between January and February, there is a dormant period for larvae that may last 30 days. Nymphs appear about 30 to 60 days after larval hatching, often during the cool, dry season from January to February. Nymphs tend to hide under the litter to prevent desiccation. Sometime from February to May they feed on the small or large mammals listed above. They molt to the adult stage within 25 to 35 days after feeding. Usually adult ticks begin to appear in June to August, during the monsoon season. Adults often hide in leaf litter but move up to the tips of grasses and plants during monsoonal showers. The period of greatest feeding activity by nymphs occurs from February to March. This is also a time when humans are actively looking for firewood within the forest and are exposed to vector ticks. Females deposit 1,000 to 2,000 eggs after engorgement.

Haemaphysalis turturis and *H. kinneari* have life cycles that are very similar to that of *H. spinigera*, though stages in their life cycles appear 4 to 6 weeks after those of *H. spinigera*. In addition, there are more overlapping broods in these species than in *H. spinigera*. Viral maintenance during the monsoon season is a potential problem in *H. spinigera*, since larvae generally do not survive the monsoon and, in any case, do not carry the virus from infected adults. Therefore, the fact that the life cycle of *H. turturis* lags behind *H.*

spinigera may be significant in maintaining the virus through the early part of the monsoon season. In addition, the seasonal life cycle of *Ixodes petauristae* is opposite that of *H. spinigera*, which may make this species even more important than *H. turturis* in maintaining the virus during the monsoon season. Larvae of *I. petauristae* peak during June to July, while nymphs peak in August and September. Except for seasonality, other aspects of the life cycles of *H. turturis* (and the other *Haemaphysalis* vector species) and *I. petauristae* are similar to *H. spinigera*. However, compared to *H. spinigera*, feeding rates on monkeys and humans are quite low. Instead, *I. petauristae* feeds primarily on shrews, rats and cattle.

Vector Surveillance and Suppression. Elimination of infected ticks would require control of ticks throughout entire forested areas. This is not technically feasible. Since many humans have become infected in the vicinity of dead monkeys, the application of an appropriate acaricide in a 50 m radius around a dead monkey has been recommended. Infected ticks do not move very far from a dead host. Military personnel should not handle dead or sick monkeys or adopt wild monkeys as pets. **Personal protective measures** discussed in TIM 36 are the most important means of preventing bites of infected ticks. An experimental vaccine has been used to prevent KFD in endemic areas of India.

K. Q Fever.

Q, or Query fever, is an acute, self-limiting, febrile rickettsial disease caused by *Coxiella burnetii*. Onset may be sudden, with chills, headache and weakness. Pneumonia is the most serious complication. There is considerable variation in severity and duration of illness. Infection may be inapparent or present as a nonspecific fever of unknown origin. Acute Q fever is self-limited, and the case fatality rate in untreated acute cases is usually less than 1%. Chronic Q fever is a serious and often fatal illness with high mortality rates. Illness occurs months to years after the acute infection, and endocarditis occurs in up to 10% of patients.

Military Impact and Historical Perspective. *Coxiella burnetii* was originally described from Australia in 1937. In ensuing years, *C. burnetii* was found to have a worldwide distribution and a complex ecology and epidemiology. Q fever first appeared among Allied troops in 1944 and 1945, when several sharp outbreaks occurred in the Mediterranean theater. The disease was not recognized immediately because this rickettsial pathogen had been reported as occurring naturally in humans only in Queensland, Australia. The need to consider Q fever in the differential diagnosis of primary atypical pneumonia was recognized during this period, but it took several years for this knowledge to become widespread in field military medicine. The British Army in the Mediterranean experienced several localized epidemics of atypical pneumonia characterized by a high attack rate, up to 50% of some units. This was probably Q fever, but no serological proof was ever obtained. Three cases of Q fever were recorded in US military personnel during the Persian Gulf War.

Disease Distribution. *Coxiella burnetii* has been reported from at least 51 countries. Incidence is greater than reported because of the mildness of many cases. Q fever is enzootic throughout South Central Asia, with most countries reporting sporadic isolated cases and occasional outbreaks. During a study in the early 1990s, over 20% of humans from the Mangla area of northern Pakistan had phase-I antibodies. High titers of phase-I antibodies to *C. burnetii* often indicates chronic infection. Serological surveys of domestic animals throughout India over the last 2 decades have demonstrated a high prevelance of *C. burnetii*. Incidence of human infection is highest in those exposed to domestic animals, such as sheep and dairy farmers, veterinary and slaughterhouse workers.

Vector Ecology Profiles. Several species of ixodid ticks transmit *C. burnetii* to animals but are not an important source of human infection. *Coxiella burnetii* has been isolated from the following species of ticks in India: *Aponomma gervaisi*, *Boophilus microplus*, *Haemaphysalis intermedia*, *H. kinneari*, *H. spinigera*, *H. turturis*, *Hyalomma hussaini*, *Rhipicephalus haemaphysaloides* and *R. sanguineus*. The role of these ticks in the epidemiology of Q fever is unknown.

Transmission Cycle(s). In nature there are 2 cycles of infection with *C. burnetii*. One involves arthropods, especially ticks, and a variety of wild vertebrates. The most important reservoirs are small wild rodents, but infection has also been demonstrated in insectivores, lagomorphs, carnivores, ungulates,

marsupials, monkeys, bats, birds, and even reptiles. *Coxiella burnetii* was isolated in India from *Aponomma gervaisi* ticks collected from a king cobra. *Coxiella burnetii* antibodies have been found in commensal rodents and shrews in India, including rats (*Rattus rattus*, *R. norvegicus*), bandicoots (*Bandicota indica*, *B. bengalensis*), the house mouse (*Mus musculus*), and the ground shrew (*Suncus murinus*). High seroprevelances of *C. burnetii* antibodies have also been found in wild brown rat populations from 4 Oxfordshire farmsteads in Great Britain. This finding is the first report of *C. burnetii* among domestic rats outside India and suggests that commensal rodents may constitute an important reservoir for this pathogen.
The other cycle is maintained among domestic animals. Although humans are rarely if ever infected by ticks, arthropods may transmit infection to domestic animals, especially sheep and cattle. Domestic animals have inapparent infections but shed large quantities of infectious organisms in their urine, milk, feces and, especially, their placental products. Because *C. burnetii* is highly resistant to desiccation, light and extremes of temperature, infectious organisms become aerosolized, causing widespread outbreaks in humans and other animals, often at a great distance from the place of origin. Dust in sheep or cattle sheds may become heavily contaminated. Once established, animal-to-animal spread of *C. burnetii* is maintained primarily through airborne transmission. Airborne particles containing rickettsiae can be carried downwind for a mile or more. Outbreaks of Q fever in humans have been traced to consumption of infected dairy products and contact with contaminated wool or hides, infected straw, and infected animal feces. *Coxiella burnetii* may enter through minor abrasions of the skin or the mucous membranes. Although rare, human-to-human transmission of Q fever has occurred. Presence of the infectious agent in the blood and tissues of patients may pose a hazard to medical and laboratory workers.

Vector Surveillance and Suppression. Although no commercial vaccine is available in the U.S., effective experimental vaccines have been developed. Severe local reactions occur in individuals with a positive skin or antibody test or a documented history of Q fever. Measures to identify and decontaminate infected areas and to vaccinate domestic animals are difficult, expensive and impractical. *Coxiella burnetii* is resistant to many disinfectants. Military personnel should avoid consumption of local dairy products and contact with domestic animals, hides or carcasses. Soldiers should not rest, sleep, or work in or near animal sheds or other areas where livestock have been housed.

L. Relapsing Fever (tick-borne).

Relapsing fever (aka: endemic relapsing fever; cave fever) is a systemic spirochetal disease characterized by periods of fever alternating with afebrile periods. The number of relapses varies from 1 to 10 or more. The severity of illness decreases with each relapse. The duration of tick-borne relapsing fever is usually longer than the closely related louse-borne relapsing fever. A number of species of *Borrelia* are responsible for the disease. The taxonomy of the pathogen is complex. The close vector-spirochete relationship has led to the definition of most spirochete species by their tick vectors. There is great strain variation among tick-borne *Borrelia*, and a single strain can give rise to many serotypes. Some authorities once viewed all species as tick-adapted strains of the louse-borne relapsing fever spirochete, *B. recurrentis*, but molecular techniques are beginning to unravel taxonomic differences between strains.

Military Impact and Historical Perspective. Although clinical symptoms of tick-borne relapsing fever can be severe, impact on military personnel would be minimal due to low incidence and the focal nature of this disease in South Central Asia.

Disease Distribution. The status of tick-borne relapsing fever is unclear in South Central Asia. Historically, sporadic cases have been reported from Afghanistan, India and Pakistan (Figure 8), and occasional cases are still reported.

Figure 8. Distribution of Tick-borne Relapsing Fever in South Central Asia.

FIG. 8. DISTRIBUTION OF TICK-BORNE RELAPSING FEVER IN SOUTH CENTRAL ASIA (DARK SHADING).

Transmission Cycle(s). Soft ticks of the genus *Ornithodoros* transmit tick-borne relapsing fever. Infection is transmitted from human to human, animal to animal, or animal to human by the bite of infective ticks. Rodents are sources of infection for ticks, although ticks are more important as a long-term reservoir. In some tick species, the pathogen has been maintained naturally for years by transovarial transmission. The rate of transovarial transmission varies greatly among tick species. Ticks of both sexes and all active stages transmit the pathogen by bite or by infectious coxal fluids exuded from pores in the basal leg segments. Spirochetes can pass into bite wounds or penetrate unbroken skin. Exposure to infected blood of patients can cause infections in medical personnel.

Vector Ecology Profiles. *Ornithodoros* spp. ticks are the vectors of tick-borne relapsing fever in South Central Asia. In addition to their role in the transmission of relapsing fever, this genus is important because it includes several species that inflict painful bites, some of which can produce local or systemic reactions in humans. Most *Ornithodoros* ticks inhabit restricted habitats, such as rock outcroppings, caves, dens, burrows, nests, and other sheltered habitats. Some species are parasitic on livestock and are found in

stables and places where host animals rest. Adult *Ornithodoros* spp. feed at night, usually for only 1 to 2 hours. Males are slightly smaller than females but similar in appearance. Larvae may remain attached to their hosts for several days. Subsequent nymphal stages are active and require blood meals in order to develop. Engorgement is rapid, and nymphs drop off their hosts after feeding. Nymphs and adults of some species feed quickly and painlessly, so their bites may go undetected by the human host until well after the tick has detached. After a variable number of molts (generally 4 to 5), adults emerge and mate. In contrast to ixodid (hard) ticks, female *Ornithodoros* do not die after oviposition. Females may live many years without a bloodmeal, but blood is required for egg development. Over the life span of the female, the number of eggs deposited may total several hundred, with up to 8 batches of eggs produced. A list of tick species and their distribution in South Central Asia appears in Appendix A.3.

Ornithodoros papillipes has been incriminated as the primary vector in central Afghanistan and infests up to 100% of wattle and daub buildings in some villages. During a survey conducted by Russian investigators in the early 1990s, *Borrelia*-infected ticks were present in 41% of infested houses, and 41 cases of relapsing fever were detected.

Ornithodoros tholozani is the most important and widespread vector in the region. It occurs across the middle of Afghanistan and through northern Pakistan and India. *Ornithodoros tholozani* tends to live in arid, sheltered areas, such as caves, rock outcroppings, huts and stables. It feeds on camels, sheep and, less frequently, man and birds. Humans are infrequent hosts of this species. Adult *Ornithodoros* spp. ticks feed at night, usually engorging in only 1 to 2 hours. *Ornithodoros tholozani* ticks also feed painlessly, so their bites may go undetected by the human host until well after the tick has detached. First instar larvae remain quiescent and do not feed, molting within a few hours. Subsequent nymphal stages are active and feed on blood. Engorgement is rapid, and nymphs drop off their hosts after feeding. After a variable number of molts, generally 4 to 5, adults emerge and mate. Females usually mate after feeding. Females may live up to 2 years without a bloodmeal, but when a host is encountered and fed upon, egg development and oviposition resume.

Vector Surveillance and Suppression. Argasid ticks like *Ornithodoros* are found in the restricted habitats of their hosts and rarely move very far. They occupy loose, dried soil of dwellings, cracks and crevices in mud-walled animal shelters, animal burrows and resting places, and the undersides of tree bark. They can be collected by passing soil through a metal sieve or by blowing a flushing agent into cracks and crevices and other hiding places. Some species are attracted by carbon dioxide, and dry ice can be used in the collection of burrow-dwelling ticks. *Ornithodoros* ticks also fluoresce under ultraviolet light. There is little seasonal fluctuation in numbers of argasids since their microhabitats are relatively stable. **Personal protective measures** (see TIM 36) are the most important means of preventing bites and diseases transmitted by soft ticks. Tents and bedding can be treated with the repellent permethrin. Encampments should not be established in areas infested with *Ornithodoros* ticks. Troops should avoid using indigenous shelters, caves, or old bunkers for bivouac sites or recreational purposes. Control of small mammals around cantonments can eliminate potential vector hosts. Rodent-proofing structures to prevent colonization by rodents and their ectoparasites is an important preventive measure. Limited area application of appropriate acaricides, especially in rodent burrows, can reduce soft tick populations. Medical personnel may elect to administer antibiotic chemoprophylaxis after exposure to tick bites when risk of acquiring infection is high. See Appendix F for **personal protective measures**.

M. Scrub Typhus.

This disease is also known as Tsutsugamushi disease, chigger-borne rickettsiosis, Japanese river fever, mite-borne typhus fever, and tropical typhus. It is a rickettsial disease characterized by a primary skin ulcer (eschar) that occurs at the site of attachment by an infected mite. The infectious agent is *Orientia* (formerly *Rickettsia*) *tsutsugamushi*. The incubation period is usually 10 to 14 days. Clinically, scrub typhus resembles other rickettsial diseases with abrupt onset of fever, headache, malaise, and swollen lymph glands. Late in the first week of fever, a maculopapular rash appears on the trunk and extends to the extremities. Without antibiotic therapy, fever lasts about 2 weeks. The case-fatality rate in untreated cases varies from 6% to 35%, but in some instances can be as high as 60% depending on the area, strain of

rickettsia, and previous exposure to the disease. Following an attack of scrub typhus, immunity to the homologous strain persists for at least 1 year. Mortality is highest among the elderly.

Historical Perspective and Military Significance. Descriptions of scrub typhus can be found in the Japanese literature as far back as 1810. The first English report of the disease appeared in 1878. Careful work during the 20 years before World War II established the basic epidemiology of the disease. Scrub typhus was second only to malaria as a cause of casualties in some Asiatic-Pacific military operations. On the Assam-Burma front, 18% of a single battalion got scrub typhus in 2 months, and 5% of the total strength died from it. The total number of scrub typhus cases in U.S. Army forces in all areas from March 1942 through December 1945 was 6,717, and 967 of these occurred in the China-Burma-India theater. Detailed understanding of scrub typhus epidemiology, effective antibiotics to treat the disease, and highly effective clothing and skin repellents should make scrub typhus less of a threat to future military operations.

Disease Distribution. Scrub typhus is widely distributed in central, eastern and Southeast Asia. It occurs from southeastern Siberia and northern Japan to northern Australia and Vanuatu, and as far west as Pakistan. It occurs as high as 3,200 m above sea level in the Himalayan Mountains. The disease is associated with a wide range of habitats, including flooded river banks, dense forests in Southeast Asia disturbed by slash and burn cultivation, semi-deserts in Pakistan, and the alpine reaches of the Himalayas. The vector mite and the pathogen typically occur in fringe habitats where 2 vegetative zones meet, such as forest and scrub. Thus, a more appropriate name for the disease is chigger-borne rickettsiosis. Historically, scrub typhus has been reported from all countries in the South Central Asian region (Figure 9), but there is little information about the number of human cases that currently occur.

Figure 9. Distribution of Scrub Typhus in South Central Asia.

Transmission Cycles. Transmission is by the bite of an infected mite of the genus *Leptotrombidium*, subgenus *Leptotrombidium*. Hosts include a variety of small mammals and birds, but the most important reservoir hosts are rats in the genus *Rattus*. However, domestic rodents are not involved in the epidemiology of scrub typhus. Only the larval stage of the mite can acquire and transmit infection, since only larvae are parasitic. Transstadial and transovarial passage of rickettsiae occur, and mites are considered the main reservoir of infection. Experimentally, it has been difficult to infect *Leptotrombidium* mites by feeding them on infected rodents, and those chiggers that become infected rarely pass the infection transovarially to offspring. Most foci of scrub typhus are the result of natural or man-made changes in the environment and are characterized by the presence of vector mites, wild rodents, particularly *Rattus* spp., and transitional secondary vegetation such as grass, shrubs and saplings.

Vector Ecology Profiles. The primary vector of scrub typhus in South Central Asia is *Leptotrombidium deliense*. This species occurs in India, Pakistan, Nepal, the Maldives, and Bangladesh. *Leptotrombidium akamushi* is another possible vector species, based on its vector status in Southeast Asia. However, this species has only been reported from India and has not been definitely incriminated as a vector in this

region. *Leptotrombidium deliense* has been found primarily in forests, while *L. akamushi* occurs mostly in grasslands.

Chigger vectors inhabit submontane, tropical, and temperate zones in the region. Hilly areas with disturbed vegetation are the primary habitats, and in many areas the life cycle continues year-round. Adult mites lay 1 to 5 eggs per day in damp, well-drained soil. Over a period of 6 to 12 weeks, 300 to 400 eggs may be deposited. The six-legged larvae that emerge from eggs ascend the tips of grasses to heights of 6 to 8 cm to await a suitable host. Most often, hosts are rodents, birds or insectivores, although humans are readily attacked. Shrews and bandicoots are common hosts in India. The distribution of the mites is dependent on the home ranges of the hosts, which do not usually overlap. Mite colonies therefore tend to be isolated from each other and occur as “mite islands.” Their focal distribution is also due to their specialized ecological requirements.

Relatively small changes in moisture content of the soil, temperature and humidity can determine survival and distribution of chigger mites. Larvae attach themselves to host tissues with mouthparts known as chelicerae and form a stylostome (combination of chigger mouthparts and host tissues, also called a feeding tube) at the point of attachment. Larvae are very small (0.15 to 0.3 mm), but after engorging they may increase sixfold in size. They are usually reddish or orange but may be pale yellow or straw-colored. On rodents or birds, primary attachment sites for larval chiggers are inside ears or around the eyes. They may also congregate around the anus and genitalia. On people, the trunk or extremities are the primary attachment sites. Chiggers seek out areas where clothing is tight against the skin, such as the waist or ankles. A lesion or eschar usually develops at the feeding site of each infected chigger.

Chiggers do not suck blood. The feeding tube allows tissues digested by the chigger’s saliva to be pumped into the digestive tract. This may require several days, although some will feed and drop off the host within 48 hours. Larvae that have fed develop into inactive, eight-legged protonymphs. Protonymphs molt to the active deutonymph stage. The next stage, the tritonymph, is also inactive and molts to the adult stage. Both the adult and deutonymph stages are free-living predators of soil-inhabiting arthropods and their eggs. Adults are small mites (1 to 2 mm), usually reddish and covered with numerous feathered hairs, giving them a velvety appearance. The nymph resembles the adult but is smaller (0.5 to 1.0 mm), and the body is less densely covered with hairs. The life cycle may occur in as little as 40 days in tropical areas, resulting in several generations per year. In more temperate areas of the Himalayas and in the semideserts of Pakistan, up to 300 or more days may be required to complete the life cycle. During cold months of the year, adults enter partial or complete hibernation.

Vector Surveillance and Suppression. Larval chiggers can be collected directly from hosts. Attached chiggers can be removed from dead or anesthetized animals with fine forceps and placed for temporary storage in 70% alcohol. Alternatively, a dead host can be placed in a jar with water and detergent, and the jar shaken vigorously to remove ectoparasites from the animal. The liquid is then poured into a funnel containing filter paper. Any mites will be strained out by the filter paper. Live hosts can be placed in cages that have wire or hardware cloth bottoms so that any mites that drop off after engorging will fall into a pan of water placed under the cage.

In the field, 12-inch squares of black paper or plastic can be placed on the ground in suspected chigger habitat for 1 to 5 minutes, after which the total number of chigger mites that congregate on the black squares are counted. Locate plates about 100 m apart. If nothing else is available, the black surface over the toes of combat boots can be used to visualize crawling chiggers. Mites can be separated from nesting material, grass, leaves and other debris with a Berlese funnel.

The wide and patchy distribution of chigger mites make their control very difficult. Vegetation can be removed mechanically or with herbicides around military encampments to make the habitat unsuitable for survival of the mites. Mite populations have been reduced by the application of residual insecticides, but this is generally not feasible over large areas. Limited applications of insecticides may be applied to the ground, vegetation and environs of camps, buildings and paths traveled by people. Permethrin-impregnated uniforms are highly effective against crawling arthropods like chiggers.

No experimental vaccine has been developed, but weekly doses of doxycycline have been shown to be an effective prophylaxis in limited studies.

N. Plague.

Plague (aka: Pestis; Black death) is a zoonotic bacterial disease involving rodents and their fleas, some species of which occasionally transmit the infection to man and other animals. The infectious agent, *Yersinia pestis,* causes fever, chills, myalgia, nausea, sore throat and headache. Bacteria accumulate and swelling develops in the lymph nodes closest to the infected bite. Since most fleabites occur on the lower extremities, the nodes in the inguinal region are involved in 90 % of cases. The term bubonic plague is derived from the swollen and tender buboes that develop. Plague is most easily treated with antibiotics in the early stages of the disease. However, untreated bubonic plague has a fatality rate of 50%. Infection may progress to septicemic plague, with dissemination of the bacteria in the bloodstream to diverse parts of the body. Secondary involvement of the lungs results in pneumonia. Pneumonic plague is of special medical significance since respiratory aerosols may serve as a source of person-to-person transmission. This can result in devastating epidemics in densely populated areas. Pneumonic or septicemic plague is invariably fatal when untreated, but responds to early antibiotic therapy. To ensure proper diagnosis, medical personnel should be aware of areas where the disease is enzootic. Plague is often misdiagnosed, especially when travelers or military personnel develop symptoms after returning from an enzootic area.

Military Impact and Historical Perspective. Epidemics of plague have been known since ancient times and have profoundly affected civilization. During the Middle Ages, Europe experienced repeated pandemics of plague. Twenty-five percent of the continent's population died during the great pandemic of the 14th century. The last pandemic originated at the close of the 19th century in northern China and spread to other continents by way of rats on steamships. During the Middle Ages, plague was a decisive factor affecting military campaigns, weakening besieged cities or attacking armies. During World War II, plague presented a real threat to U.S. military forces in the Mediterranean area and the Orient, but no U.S. military personnel contracted the disease. This was attributed to effective rodent control, DDT for flea control, chemoprophylaxis, and the use of preliminary plague vaccines. Severe ecological disturbances and dislocations of human populations during the Vietnam War led to outbreaks of plague, primarily in native populations. Even though plague has been declining on a worldwide basis, persistent enzootic foci can trigger the recurrence of epidemics when war or natural disaster disrupts general sanitation and health services. Presently, the threat of plague to military operations is low.

Disease Distribution. See Figure 10.

Figure 10. Probable Foci of Plague in South Central Asia.

Afghanistan: The last human cases were officially reported in the 1970s, but plague is probably still enzootic in gerbils.

India: Plague has been present in India for centuries, although the first definite record was in 1896. Between 1896 and 1914, 183,984 deaths were attributed to plague in Bombay, and in India overall the mortality amounted to 8,538,931. During most of the twentieth century, cases diminished steadily until only 678 were reported from 1959 to 1968. Until the plague epidemic of 1994, the last official cases in humans were in 1967. However, a suspected outbreak of pneumonic plague occurred in 1983 in a small village of Himachal Pradesh, where 17 of 22 affected persons died.

On August, 26, 1994, cases of bubonic plague appeared in 15 villages in Beed District, Maharashtra State, and by September 24, over 450 cases had been reported with 41 deaths. A second outbreak, which was suspected to be pneumonic plague, was reported in the industrial city of Surat, Gujarat State, on September 22. From August 26 to October 5, a total of 5,150 suspected pneumonic or bubonic plague cases were

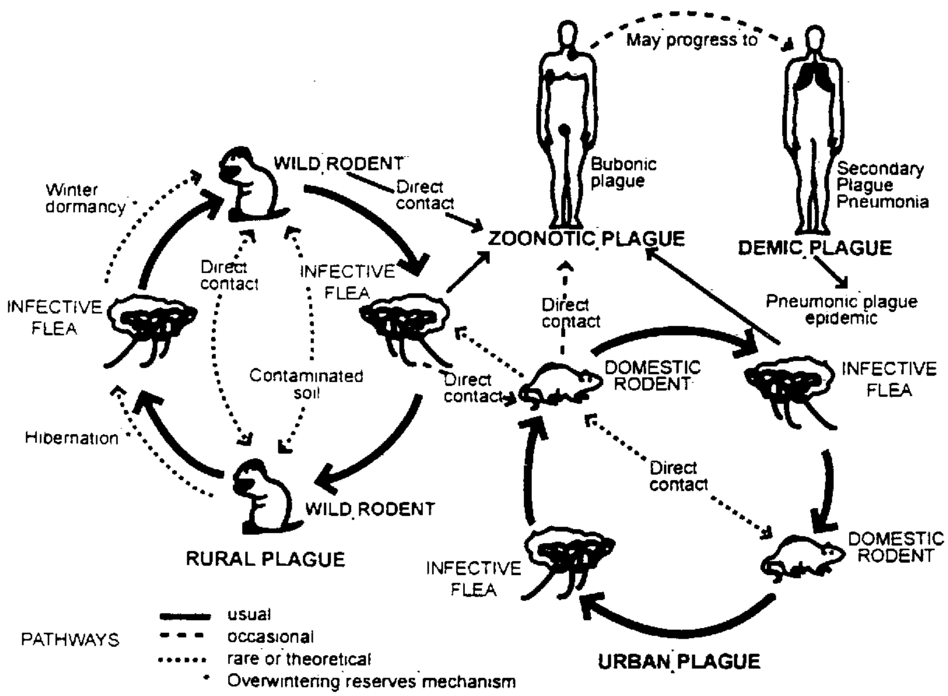
reported from eight states of India, primarily in the south-central and southwestern regions. Of these, 2,793 (54.2%) were reported from Maharashtra State (including Bombay), 1,391 (27.0%) from Gujarat State (including the city of Surat), 749 (14.5%) from Delhi, and 169 (3.3%) from the states of Andhra Pradesh, Haryana, Madhya Pradesh, Rajasthan, Uttar Pradesh, and West Bengal (including Calcutta). Only a small proportion of suspected cases were microbiologically confirmed. Living conditions in parts of Surat were appalling, and slums were heavily infested with rats. Several important lessons were learned from this epidemic. One was the archaic state of the public health infrastructure in India. The second was the fear and panic that spread among the people and media during the epidemic. An estimated 500,000 people fled the city of Surat and the surrounding areas, including doctors and other medical personnel. People started wearing masks in the streets, not only in Surat but in Bombay, New Delhi, and other cities hundreds of kilometers away. Some nations felt compelled to restrict trade and travel with India. Tourism declined by 50%. It has been estimated that the epidemic cost the Indian economy more than 2 billion dollars in lost trade and tourism. Lastly, it became apparent that little is known about the current status of enzootic plague in India.

Nepal: The last human cases of plague were reported in 1967, 5 km from Nawra in the northwestern part of Nepal, near the Tibetan border. *Pulex irritans* was the flea believed to be involved in transmission of plague to humans. No evidence of a rodent epizootic was found in the area where the outbreak occurred, and the enzootic status of plague is still unclear in Nepal. Nepal is believed to represent part of the southernmost boundaryof the ancient plague focus of Central Asia, to which the old plague foci in the Indian provinces of Garhwal and Kumaon belong.

Transmission Cycle(s). Plague is a disease of rodents. It is maintained in nature among wild rodents and their fleas (Figure 11). This zoonotic cycle is termed sylvatic, campestral, rural, or wild plague and can be very complex, involving many rodent and flea species. Worldwide, over 220 species of rodents have been shown to harbor *Y. pestis*. In addition, the camel and goat are susceptible to infection with plague bacteria and may have a significant role in the dissemination of human plague when infected animals are butchered for human consumption.

Figure 11. Epidemiological Cycles of Plague.

FIGURE 11. EPIDEMIOLOGICAL CYCLES OF PLAGUE



Some rodents are highly susceptible to infection, resulting in high mortality. Although large numbers of dead and dying rodents are a good indication of an epizootic of plague, rodent species that are resistant to infection are more important in maintaining the zoonotic cycle. Most cases in military personnel would probably occur as a result of intrusion into the zoonotic cycle during or following an epizootic of plague in wild rodents. Domestic cats and dogs may carry infected rodent fleas into buildings or tents. Cats may occasionally transmit infection by their bites or scratches, or by aerosol when they have pneumonic plague. Troops should not be allowed to adopt cats or dogs as pets during military operations.

The entry of wild rodents or their infected fleas into human habitations can initiate an epizootic among commensal rodents, primarily *Rattus* spp., which are highly susceptible to infection. Close association of humans and large populations of infected commensal rodents can result in an urban cycle of plague. A similar cycle can occur in military cantonments experiencing large infestations of commensal rodents. The most important vector of urban plague worldwide is the Oriental rat flea, *Xenopsylla cheopis*. The northern rat flea, *Nosopsyllus fasciatus*, and the human flea, *Pulex irritans*, are secondary vectors. A complete list of South Central Asian flea species and their distributions appears in Appendix A.4.

Plague is transmitted to humans primarily by the bite of infected fleas. Fleas often exhibit a host preference, but most species of medical importance readily pass from one host to another. A lack of absolute host specificity increases the potential for infection and transmission of pathogens. Plague may also be acquired by handling tissues of infected animals or humans, and by person-to-person transmission of pneumonic plague. Crushed infected fleas and flea feces inoculated into skin abrasions or mucous membranes can also cause infection. Not all flea species are competent vectors. The vector competence of the Oriental rat flea is attributed to enzymes produced by the plague bacilli that cause blood to coagulate in the flea's digestive tract. The flea attempts to clear the blockage in its digestive tract by repeated efforts to feed. In the process, plague bacilli are inoculated into the host. Fleas may remain infective for months when temperature and humidity are favorable. *Xenopsylla cheopis* may require 2 to 3 weeks after an infective blood meal before it can transmit plague bacilli.

Some of the more important wild rodents that maintain the enzootic cycle of plague in India include susliks, ground squirrels, jerboas, wood rats, striped mice, Asiatic striped palm squirrels (*Funambulus* spp.) and gerbils, especially *Tatera indica*. Peridomestic rodents such as the house shrew, *Suncus murinus*, and bandicoot rats, *Bandicota bengalensis* and *B. indica*, that mix freely with domestic rodents, such as *Rattus rattus* and *R. norvegicus*, may be important in linking the rural and urban cycles of plague.

Vector Ecology Profiles. In India, the primary epidemic plague vectors are *Xenopsylla cheopis* (the Oriental rat flea) and *X. astia*, though *X. cheopis* is generally considered to be the better vector. In many parts of India, *X. cheopis*, which is an introduced species, is replacing *X. astia*, the indigenous species. The distribution of these vectors is largely determined by the distribution of their hosts, primarily *Rattus rattus*, *R. norvegicus*, *Mus musculus*, *Tatera indica*, *Suncus murinus*, *Bandicota bengalensis*, and *B. indica*. Both *X. cheopis* and *X. astia* were found in states with recent plague outbreaks, including Maharashtra, Gujarat, Uttar Pradesh, and Himachal Pradesh. The states with the highest flea indices (number of fleas per rodent host) were Maharashstra, Uttar Pradesh, and Himachal Pradesh. Within these states, most areas had flea indices > 1.0, indicating that plague could easily be transmitted from rat to rat. These 2 flea species are widely distributed in virtually every Indian state. They also occur in Pakistan, Afghanistan, and Nepal. In some areas, particularly West Bengal, flea indices have been gradually decreasing because of statewide campaigns to eradicate rats. As rat populations have declined, they are largely replaced by bandicoots, with the result that the proportion of *X. astia* has risen relative to *X. cheopis*. *Xenopyslla astia* indices increase from December to March, reaching a peak in April to May. Flea populations decline precipitously during the monsoon season from June to September, and remain low through November.

Xenopsylla cheopis occurs mostly in urban areas, in association with its rodent hosts. However, it may occur sporadically in villages when rats are present. Adult fleas feed exclusively on blood and utilize blood protein for egg production. After feeding on a rodent, the female Oriental rat flea lays several eggs (2 to 15). Hundreds of eggs may be laid during the entire life span. Oviposition most often occurs on the hairs of the host, but the eggs drop off and hatch in the nest or its environs. In locally humid environments, such

as rodent burrows, eggs may hatch in as little as 2 days. Larvae grow rapidly when temperature and humidity are above 25°C and 70% R.H., living in the nest and feeding on dried blood, dander, and other organic materials. The larval stages can be completed in as little as 14 days (at 30 to 32°C), or as long as 200 days when temperatures drop below 15°C or when nutrition is inadequate. Mature larvae pupate in cocoons, loosely attached to nesting material. Adult emergence may occur in as little as 7 days or as long as a year and is stimulated by carbon dioxide or host activity near the cocoon. Adult fleas normally await the approach of a host rather than actively search for one. Fleas feed on humans when people and rodents live close together, but man is not a preferred host. However, if rat populations decline suddenly due to disease or rat control programs, these fleas readily switch to feeding on humans. The life span of adult *X. cheopis* is relatively short compared to that of other flea species, often less than 40 days. Flea populations increase rapidly during periods of warm, moist weather.

The life cycle of *X. astia* is very similar to that of the Oriental rat flea except that it lives on both wild and commensal rodents. It occurs widely in southern India, Pakistan, Bangladesh, and Sri Lanka. Bandicoots and shrews are the preferred hosts in rural areas. Its seasonal peaks generally coincide with those of *X. cheopis*.

Pulex irritans, commonly termed the human flea, occurs mainly among lower socioeconomic groups. It is a secondary vector of plague in the region. The life cycle of the human flea is similar to that of the Oriental rat flea. Despite its common name, *P. irritans* has a low to moderate preference for humans and is more likely to feed on rats, mice and gerbils, maintaining the enzootic plague cycle among these hosts. Where swine occur, the human flea prefers this host to humans. Domestic animals such as dogs also serve as hosts but, in the absence of preferred hosts, this flea readily feeds on humans and is frequently found in human habitations. *Pulex irritans* can live over one year on its preferred hosts. It can survive unfed for several months.

The northern rat flea, *Nosopsyllus fasciatus*, is involved only in the sylvatic plague transmission cycle. This species is rare in arid areas of the subcontinent but is otherwise widely distributed. *Nosopsyllus fasciatus* has a life cycle similar to that of other fleas and lays its eggs in the nests or burrows of commensal rodents. Larvae have the unique habit of attaching to the abdomen of an adult flea and ingesting fecal blood as it passes from the anus of the adult. Adults of this species rarely feed on man. It is tolerant of a broader range of temperatures than the Oriental rat flea but prefers cooler temperatures. Under favorable conditions, the adult life span is just under 100 days. Adults of *N. fasciatus* can survive unfed for several months.

Vector Surveillance and Suppression. The methods of flea surveillance depend upon the species of flea, the host, the ecological situation, and the objective of the investigation. Fleas can be collected from hosts or their habitat. The relationship of host density to flea density should be considered in assessing flea populations. It has been common practice for years to use a flea index (average number of fleas per host), especially in studies of rodent fleas. For *X. cheopis*, a flea index > 1.0 flea per host is considered high. The flea index has many limitations, since only adults are considered and then only while they are on the host. Fleas are recovered by combing or brushing the host or by running a stream of carbon dioxide through the fur while holding the host over a white surface.

Flea abundance in the environment can be determined by counting the number of fleas landing or crawling in 1 minute on the lower parts of the legs of the observer. The trouser legs should be tucked into the socks to prevent bites. Flea populations can also be estimated by placing a white cloth on the floor in buildings or on the ground in rodent habitat and counting the fleas that jump onto the cloth. Various flea traps have been devised. Some use light or carbon dioxide as an attractant. Sifting and flotation of rodent nesting materials or of dust and debris from infested buildings are effective methods of collecting fleas from the environment.

Serologies of wild carnivores are sensitive indicators of enzootic plague. Fleas and tissues from suspected reservoirs or humans may be submitted for plague analysis to the Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Vector-borne Infectious Diseases, P.O. Box 2087, Foothills Campus, Fort Collins, Colorado 80522. Blood samples are easily collected on Nobuto

paper strips, dried and submitted to a laboratory for testing. Consult TG 103, Prevention and Control of Plague.

Control of enzootic plague over large areas is not feasible. Control efforts should be limited to foci adjacent to urban areas, military encampments, or other areas frequented by military personnel. If possible, cantonment sites should not be located in wild rodent habitat. Fleas quickly leave the bodies of dead or dying rodents in search of new hosts. Consequently, flea control must always precede or coincide with rodent control operations. Application of insecticidal dusts to rodent burrows is effective in reducing flea populations, but it is very labor intensive. Fleas can be controlled by attracting infested rodents to bait stations. The stations may incorporate an insecticidal dust that rodents pick up while feeding or a rodent bait containing a systemic insecticide that fleas ingest when taking a bloodmeal. However, baiting with systemic formulations may pose environmental risks.

Urban plague control requires that rodent runs, harborages and burrows be dusted with an insecticide labeled for flea control and known to be effective against local fleas. Insecticide bait stations can also be used. Rat populations should be suppressed by well-planned and intensive campaigns of poisoning and concurrent measures to reduce rat harborages and food sources. Buildings should be rat-proofed to the extent possible to prevent rats from gaining entry. Domestic rodent control is discussed in Technical Guide (TG) 138, Guide to Commensal Rodent Control. Insecticides recommended for flea control are listed in TIM 24, Contingency Pest Management Pocket Guide.

Military personnel, especially those involved in rodent control, should use the **personal protective measures** discussed in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance, and outlined in Appendix F. Active immunization with a vaccine of killed bacteria confers protection against bubonic plague (but not pneumonic plague) in most recipients for several months. Booster injections are necessary every 6 months. The efficacy of plague vaccine in humans has not been demonstrated in a controlled trial, so vaccination should not be relied upon as the sole preventive measure.

O. Murine Typhus.

The infectious agent, *Rickettsia typhi*, causes a debilitating illness with high fever and a maculo-papular rash that is also referred to as flea-borne typhus, endemic typhus, and shop typhus. The incubation period ranges from 1 to 2 weeks, and clinical symptoms may last up to 2 weeks in untreated cases. Mortality is very low, and serious complications are infrequent. The disease is easily treated with antibiotics. Absence of louse infestation, seasonal distribution, and the sporadic occurrence of murine typhus help to differentiate it from epidemic typhus. Murine typhus is often unrecognized and substantially underreported in most endemic areas.

Military Impact and Historical Perspective. Confusion in diagnosis between murine typhus and closely related diseases may occur. Prior to World War II, murine typhus was not distinguished from the epidemic form, and its importance in prior wars is unknown. During World War II, there were 786 cases in the US Army, with 15 deaths. Only 34 cases were recorded in the China-Burma-India theater. There are little available data on the incidence of murine typhus during military operations in Korea or Vietnam. During the Vietnam War, murine typhus was concentrated in port cities and incidence seemed low. However, retrospective studies indicated that a large proportion of fevers of unknown origin experienced by Americans during that conflict were probably due to *R. typhi*. The disease is most common in lower socioeconomic classes and increases when wartime disruptions or mass migrations force people to live in unsanitary conditions in close association with domestic rodents. However, murine typhus has not been a major contributor to disease rates in disaster situations. Because of the sporadic incidence of murine typhus, it is difficult to confidently predict the potential impact of this disease on future military operations, although any such impact would probably be minimal.

Disease Distribution. Murine typhus is one of the most widely distributed arthropod-borne infections and is endemic in many coastal areas and ports throughout the world. Human cases occur principally in urban areas, where commensal rodent infestations are common, although infected rodents have been collected

from rural villages. Sporadic cases are reported from South Central Asia. Foci are countrywide wherever rats are common, and transmission is year-round in tropical and subtropical regions. Eight cases were reported from Sri Lanka in 1993. All 8 patients lived in rat- infested houses. Murine typhus usually occurs in the summer and fall, while epidemic typhus generally occurs during colder months.

Transmission Cycle(s). Murine typhus is a zoonotic infection associated with domestic rats (*Rattus rattus* and *R. norvegicus*) and vectored by their fleas and the spiny rat louse, *Polyplax spinulosa*. The Oriental rat flea, *X. cheopis*, is the most important vector. Neither rodents nor their ectoparasites are affected by infection with *R. typhi*. Inoculating crushed fleas or infective flea feces into the skin at the bite site transmits murine typhus. Scratching fleabites increases the likelihood of infection, but *R. typhi* is rarely transmitted directly by fleabite. Other routes of infection are inhalation of dry flea feces containing rickettsiae (which may remain infective for months), and ingestion of food contaminated by rodent urine. Murine typhus is not transmitted from person to person. The risk of transmission is year-round but peaks during the warm months in northern areas of South Central Asia.

Although the rat-flea-rat cycle is still the major source of human infection, murine typhus exists in some endemic foci where commensal rodents are absent. In suburban areas of Texas and southern California, the classic enzootic cycle has been replaced by a peridomestic animal cycle involving free-ranging cats, dogs and opossums and their fleas. In the Dinaric beech-fir forest of southern Slovenia, *Monopsyllus sciurorum* fleas collected from the nests of the fat doormouse, *Glis glis*, were found infected with *R. typhi*. The widespread distribution of this sylvatic flea species and its presence on other mammalian and avian hosts suggests that murine typhus may exist in unrecognized enzootic cycles.

Vector Ecology Profiles. The primary vectors are the Oriental rat flea, *X. cheopis*, and *X. astia*. Cat and dog fleas, *Ctenocephalides felis* and *C. canis*, as well as the human body louse, *Pediculus h. humanus*, are potential secondary vectors for humans. The northern rat flea, *Nosopsyllus fasciatus*, spiny rat louse, *Polyplax spinulosa*, and tropical rat mite, *Ornithonyssus bacoti*, are vectors that maintain the enzootic cycle of the disease. The fleas of South Central Asia and their distribution are listed in Appendix A.4. Flea biology is discussed under plague. The biology of human body lice is discussed under epidemic typhus.

Polyplax spinulosa, the spiny rat louse, is closely associated with its rodent hosts. Female lice attach eggs to rodent hairs, and all developmental stages live exclusively on rodents. Lice are only transferred from rodent to rodent by body contact. These lice feed on rat blood but do not feed on humans.

Ornithonyssus bacoti, the tropical rat mite, lives on commensal and other rodents throughout the region and feeds on blood and other fluids that ooze from its tiny bite wounds. Engorged females start laying eggs within 2 days of feeding and continue to lay groups of eggs for 2 to 3 days. Eggs hatch in 1 to 2 days and develop into larvae, followed by protonymphs and deutonymphs. The entire life cycle, from egg to adult, requires only 5 to 6 days. These mites will readily infest humans if their rodent hosts are suddenly eliminated, or if humans live in close association with rodent nests.

Vector Surveillance and Suppression. See plague.

P. Epidemic Typhus.

Epidemic typhus is a severe disease transmitted by the human body louse, *Pediculus humanus humanus*. The infectious agent is the bacterium *Rickettsia prowazekii*. It causes high mortality, particularly in populations weakened by malnutrition. Case fatality rates normally vary from 10 to 40% in the absence of specific therapy. Onset is usually sudden and marked by fever, headache, and general pains followed by a rash that spreads from the trunk to the entire body. Untreated cases of epidemic typhus may last up to 3 weeks. Many humans who contract typhus retain some rickettsiae for the rest of their lives. Under certain stressful conditions or reduced immunity, they may relapse and develop a milder form of typhus known as Brill-Zinsser disease.

Military Impact and Historical Perspective. Epidemics of typhus have changed the course of history. One author has stated that the louse has killed more soldiers than all the bullets fired during conflict. In one of the worst disasters in military history, over half of Napoleon's army perished from epidemic typhus during the invasion of Russia in 1812. During the first year of World War I, typhus started as an epidemic in the Serbian Army. In 6 months, 150,000 people had died of the disease, including 50,000 prisoners of war and one-third of Serbian physicians. By the end of the war and during the period immediately following it (1917 to 1923), an estimated 30 million cases of epidemic typhus occurred in Russia and Europe, with over 300,000 deaths. During World War II, there were severe epidemics of typhus in some endemic areas. Large epidemics occurred in Bucovina, northeast Romania, and neighboring Moldova. There were hundreds of thousands of cases in Poland during the war as well as large epidemics in Yugoslavia. From 1941 to 1944, there were over 132,000 cases in urban areas of French North Africa. Over 91,000 cases occurred in Egypt during the same period. Despite this incidence, US Army personnel experienced only 30 cases of typhus with no typhus deaths in the North African-Middle East-Mediterranean zone during the years 1942 to 1945. When Allied forces landed in Italy in 1943, a typhus epidemic in Naples was ravaging the city of 1 million. Death rates reached 80%. An effective delousing campaign, chiefly using DDT, was waged. This marked the first time in history that an epidemic of typhus did not exhaust itself but instead was terminated by human action. The U.S. Army achieved a remarkable record of low morbidity with no fatalities from epidemic typhus in World War II by taking effective protective measures against the disease and through the work of the US Typhus Commission established by the Secretary of War on October 22, 1942.

The development of modern antibiotics and insecticides has reduced the threat of this disease to military forces. However, the short incubation period and severe clinical symptoms of epidemic typhus should be of concern to medical personnel when dealing with large concentrations of refugees and prisoners of war. *Rickettsia prowazekii* has the most serious epidemic potential of all rickettsiae, and the emergence and dissemination of body lice can be very rapid under favorable conditions. In a refugee camp in Goma, Zaire, all 800,000 refugees became infested within 1 month.

Disease Distribution. Epidemic typhus is more common in temperate regions and in the cooler tropics above 1,600 m. It is absent from lowland tropics. It usually occurs in mountainous regions where heavy clothing is worn continuously, such as the Himalayas, Pakistan and Afghanistan, and the highlands of Ethiopia. The incidence of epidemic typhus has been steadily declining in the last 2 decades. The majority of recent cases have occurred in Africa, primarily in Ethiopia, with most of the remainder occurring in Peru and Ecuador. During 1997, a large outbreak was reported in Burundi in which 100,000 people were infected. A small outbreak was observed in Russia in the same year. During 1998, small outbreaks were recorded from Peru, and an isolated case occurred in Algeria. Epidemic typhus may still be endemic in rural highlands of South Central Asia, although there are no published reports of recent cases. Up to 13% of surveyed adults in Afghanistan have been seropositive, and cases of epidemic typhus were reported in Afghan refugee camps along the western border of Pakistan during the 1980s. Body louse infestations can be very high in many areas of the region. A 1989 survey of the population of Shillong, a hilly city in northeast India situated at 1,500 m elevation, found that 30.6% of the mostly poor people living in the suburban areas were infested with body lice, while 6.4% of the educated middle-class people living in the urban parts of the city were infested.

Transmission cycle(s). The head louse, *Pediculus humanus capitis*, and the crab louse, *Pthirus pubis*, can transmit *R. prowazekii* experimentally, but epidemics have always been associated with the body louse, *P. h. humanus*. Humans are reservoirs of the pathogen and the only hosts for the lice. Transmission of the disease occurs when individuals wear the same clothes continuously under crowded, unsanitary conditions. Major epidemics have been associated with war, poverty and natural disasters. Persons in cold climates are more likely to acquire epidemic typhus when they are unable to bathe or change clothes for long periods of time.

Lice become infective 5 to 7 days after a blood meal from an infected human. During subsequent blood meals, the louse defecates and rickettsiae are excreted in the feces. Louse bites are irritating, and scratching by the host produces minor skin abrasions, which facilitate entry of the pathogen from feces or

crushed body lice. *Rickettsia prowazekii* can survive desiccation for several weeks. Louse feces are extremely dry and powdery, so infection may also occur by inhalation of infective louse feces.

The survival of *R. prowazekii* between outbreaks is of interest, since there is no transovarial transmission and lice die from the infection. Individuals who recover from the initial infection and relapse years later with Brill-Zinsser disease are considered the primary reservoir. Lice feeding on such patients become infected. A sylvatic cycle of *R. prowazekii* has been recognized in the southeastern United States, where flying squirrels and their ectoparasites (the flea *Orchopeas howardii* and the louse *Neohaematopinus sciuropteri*) are naturally infected. The louse is host specific, but *O. howardii* has an extensive host range, which includes humans. Sporadic human cases have occurred in houses harboring flying squirrels. The significance of this finding to the epidemiology of epidemic typhus in other areas is not known.

Vector Ecology Profiles. Human lice spend their entire life cycle (egg, 3 nymphal stages and adult) on the host. Eggs of body lice are attached to clothing at a rate of about 5 to 8 eggs per female per day. Lice must mate before egg laying, since females cannot store sperm. At 29 ^{o}C to 32 ^{o}C, eggs hatch in 7 to 10 days. The maximum time eggs can survive unhatched is 3 to 4 weeks, which is important when considering the survival of lice in infested clothing and bedding. A bloodmeal is required for each of the 3 nymphal molts and for egg production in adults. The nymphal stages are passed in 8 to 16 days. Louse populations have the potential to double every 7 days. Adults live about 2 weeks and feed daily. Infestations of lice cause considerable irritation and scratching, which may lead to skin lesions and secondary infections. Body lice are commonly found in the seams and folds of clothing. Lice tolerate only a narrow temperature range and will abandon a dead host or one with a body temperature of 40 ^{o}C or above. This contributes to the spread of lice and louse-borne disease. Humidity is also critical because lice are susceptible to rapid dehydration. The optimal humidity for survival is between 70% and 90%. They cannot survive at a relative humidity below 40%. Human lice can survive without a host for only a few days and are spread by intimate personal contact or contact with infested clothing or other items.

Vector Surveillance and Suppression. The incidence of head lice has been increasing worldwide. Body louse infestations have declined with higher standards of living, although infestations are still common in some areas of South Central Asia, especially at high elevations where heavy clothing is worn and bathing is infrequent. The prevalence of body lice reflects the socioeconomic level of the society. The incidence of body lice has increased in some countries due to war and social change. Infestations with body lice are increasingly being reported among the homeless and deprived populations in inner cities of developed nations. Military personnel should avoid close personal contact with infested persons and their belongings, especially clothing and bedding.

Surveillance for body lice consists of examining individuals and their clothing for lice or nits (eggs). The population density of body lice may be very high, but usually only a few lice are observed on an individual. Body lice are frequently found around the waistbands of clothing. Heavily bitten areas, such as the base of the thorax, the groin and the flanks of the body, may become darker. This characteristic skin coloration is often referred to as vagabond's disease.

Dry cleaning or laundering clothing or bedding in hot water (55 ^{o}C for 20 minutes) will kill eggs and lice. Control of epidemics requires mass treatment of individuals and their clothing with effective insecticides. The permethrin-treated BDU is extremely effective against lice. Since lice cannot survive away from the human host, application of insecticides to buildings, barracks or other living quarters is not necessary. Mass louse control could be hampered by insecticide resistance. Resistance to common pediculicides, particularly DDT and gamma BHC (lindane), is widespread in South Central Asia. Pyrethroid lotions and shampoos have been widely used in this region to control head lice, and reports of pyrethroid resistance are increasing. Compounds such as ivermectin, taken orally to eradicate lice, have been investigated experimentally but are not currently registered for that use. Additional information on the surveillance and controlof body lice can be found in TIM 6, Delousing procedures for the control of louse-borne disease during contingency operations.

Production of typhus vaccine in the United States has been discontinued, and there are no plans for commercial production of a new vaccine. Vaccination against typhus is not required by any country as a

condition of entry. The U.S. military no longer has the equipment to perform mass delousing as it has done in the past.

Q. Relapsing Fever (louse-borne).

Epidemic louse-borne relapsing fever is caused by the spirochete *Borrelia recurrentis*. The symptoms and severity of relapsing fever depend on the immune status of the individual, geographic location, and strain of *Borrelia*. The incubation period in an infected host ranges from 2 to 14 days. The disease is characterized by a primary febrile attack followed by an afebrile interval and 1 or more subsequent attacks of fever and headache. Intervals between attacks range from 5 to 9 days. In untreated cases, mortality is usually low but can reach 40%. Infection responds well to treatment with antibiotics.

Military Impact and Historical Perspective. Major epidemics of louse-borne relapsing fever occurred during and after World War I in Russia, Central Europe and North Africa. After the war, relapsing fever was disseminated through large areas of Europe, carried by louse-infested soldiers, civilians and prisoners of war. Between 1910 and 1945, there were an estimated 15 million cases and nearly 5 million deaths. Large outbreaks of relapsing fever were common during and after World War II, when epidemics in North Africa produced an estimated 1 million cases and some 50,000 deaths. However, U.S. forces were largely spared. There were only 70 cases reported from the China-Burma-India theater and most of these occurred in China. During the Vietnam War, outbreaks of louse-borne relapsing fever occurred in the Democratic People's Republic of Vietnam.

Disease Distribution. From 1960 to 1980, louse-borne relapsing fever flourished primarily in the Sudan, Somalia, Ethiopia and Eritrea. Ethiopia reported the highest number of cases, with an estimated 10,000 per year. Relapsing fever is also believed to persist in the Peruvian Andes and the Himalayas. Epidemics usually occur in the cold season, among poor people with inadequate hygiene. There are no recent published reports of louse-borne relapsing fever in South Central Asia.

Transmission Cycle(s). The body louse, *P. h. humanus*, is the vector of *B. recurrentis*. After the louse feeds on infective blood, the spirochetes leave the digestive tract and multiply in the insect's body cavity and other organs. They do not invade the salivary glands or the ovaries and are not found in the feces. Bites and fecal deposits cannot transmit the pathogen, and transovarial transmission does not occur. Human infection occurs when a louse is crushed and *Borrelia* spirochetes are released. The spirochetes may be scratched into the skin or come in contact with mucous membranes, but there is evidence that *B. recurrentis* can penetrate unbroken skin. Since infection is fatal to the louse, a single louse can infect only 1 person. However, *B. recurrentis* can survive for some time in a dead louse. Outbreaks of louse-borne relapsing fever require high populations of body lice. Lice leave febrile patients in search of new hosts, and this behavior contributes to the spread of disease during an epidemic.

Humans are the only known reservoir for *B. recurrentis*. Mechanisms of survival during non-epidemic periods are unknown. The life cycle of the body louse is less than 2 months, and in the absence of transovarial transmission *B. recurrentis* cannot survive in the louse population.

Vector Ecology Profiles. See epidemic typhus.

Vector Surveillance and Suppression. See epidemic typhus. Also consult TIM 6. Delousing procedures for the control of louse-borne disease during contingency operations.

R. Other Arthropod-borne Viruses.

Many enzootic arboviruses are circulating in South Central Asia, but little is known about them. Available epidemiological information indicates that they would have a minor impact on military operations. However, medical personnel should be aware of these arboviruses because they will frequently be treating fevers of unknown origin and, in serological tests, may see reactions to closely related viruses known to cause human disease in the region.

Bhanja viral infection is a tick-borne disease appearing as a simple febrile illness or with symptoms of meningitis. This pathogen is an unclassified virus in the family Bunyaviridae. Bhanja virus was first isolated in 1954 from *Haemaphysalis* ticks collected on goats in India. It is unlikely to have a significant military impact due to the high percentage of asymptomatic infections and the mild nature of the clinical disease.
Bhanja virus has been isolated from at least 15 countries of Asia, Africa and Europe, and serological evidence indicates it may be present in at least 15 additional countries. The virus has been isolated from ticks in India, Pakistan and Sri Lanka. Isolation of Bhanja virus from mammals is rare. The virus has been isolated from *Haemaphysalis intermedia* in Orissa, India. Antibodies to Bhanja virus are common in domestic animals and have occasionally been found in rodents and birds. A characteristic feature common to all natural foci of the virus is intense pasturage of cattle, sheep or goats and abundant ticks of the genera *Dermacentor* and *Haemaphysalis*. **Personal protective measures** (see TIM 36) afford the best protection against the ticks that transmit Bhanja virus. Military personnel should be especially vigilant in areas used to graze or hold sheep and goats.

Human infection with Batai virus (*Bunyavirus*, Bunyaviridae) produces a fever and occasionally a meningitis syndrome. Batai virus has been isolated from pigs in India and from mosquitoes in Sri Lanka. *Culex bitaeniorhynchus*, *Anopheles barbirostris*, *An. subpictus*, and *An. tessellatus* are potential vectors of Batai virus in the region. Domestic ungulates have a high prevalence of Batai antibodies in India. Batai virus is the only Asian representative of the Bunyamwera group of the Bunyaviridae.

Getah virus (*Alphavirus*, Togaviridae) has caused illness in horses in India and has been isolated from *Cx. gelidus*, *Cx. fuscocephala* and *Cx. tritaeniorhychus* collected in the vicinity of swine in Sri Lanka. This virus has been implicated in stillbirth and abortion in pigs, but infection in cattle is benign. The natural transmission cycle of this alphavirus is not known, but is thought to be similar to the epidemiology of JE with pigs acting as an amplifying host. Antibodies to Getah virus without human disease have been reported in several South Central Asian countries, and it is not considered a public health threat.

Chandipura virus (*Vesiculovirus*, Rhadoviridae) is transmitted by phlebotomine sand flies and infects domestic animals in India. It is known to cause a mild but rare febrile illness in humans. Antibodies to Chandipura virus have been reported from wild toque macaques (*Macaca sinica*) in Sri Lanka. This virus has been experimentally transmitted by *Aedes aegypti* in the laboratory.

Dera Ghazi Khan virus (*Nairovirus*, Bunyaviridae) infects camel ticks, *Hyalomma dromedarii*, in Pakistan. Little is known of vertebrate hosts or natural cycles of infection. Antibodies to this virus may cross-react with the closely related Crimean-Congo hemorrhagic fever virus.

Antibodies to Vellore virus (*Orbivirus*, Reoviridae) have been detected in cattle and humans in India, though no human disease has been associated with infection. The virus has been isolated from *Culex pseudovishnui* in India.

Wanowrie virus is an ungrouped virus closely relate to *Bunyavirus*, Bunyaviridae. It was first isolated from ticks, *Hyalomma marginatum isacci* and subsequently from a human febrile case in India. A serosurvey of 100 human sera randomly selected from 6 states (Jammu and Kashmir, Rajasthan, Orissa, Maharashtra, Karnataka, and Tamil Nadu) found antibodies to Wanowrie virus in 15% of the sera. Wanowrie virus was isolated from the brain of a fatal human case of encephalitis in Sri Lanka.

Dhori virus in the family Orthomyxoviridae has been isolated from *Hyalomma* ticks collected from domestic ruminants in India. It has been associated with febrile illness and signs of central nervous system involvement in workers infected in the laboratory.

Umbre virus (*Bunyavirus*, Bunyaviridae) naturally infects *Culex* mosquitoes, especially *Cx. vishnui*, *Cx. bitaeniorhynchus* and *Cx. pseudovishnui*. The virus has been isolated from wild birds and chickens in India. No human disease is recognized, but neutralizing antibodies to Umbre virus are prevalent in humans tested in Malaysia.

VI. Militarily Important Vector-borne Diseases with Long Incubation Periods (>15 days)

A. Leishmaniasis.

This potentially disfiguring and sometimes fatal disease is caused by infection with protozoan parasites of the genus *Leishmania*. Transmission results from bites of infected phlebotomine sand flies. The parasite responsible for The Black Sickness (Kala-azar) or Dum Dum Fever (named for the Indian city of Dum Dum) was described in 1903 by William Leishman on the basis of a case in a British soldier assigned to Dum Dum, India. Also in 1903, Charles Donovan reported the same parasite from a splenic puncture of a British soldier, who had been assigned to troop duty in India and was sick with the Black Sickness. The first identification of an infected sand fly, and the first proven transmission of the parasite by the bite of the sand fly vector *Phlebotomus argentipes*, were accomplished by military medical personnel in India. All vectors of leishmaniasis in the Old World belong to the sand fly genus *Phlebotomus*. Incubation in humans may take as little as 10 days or more than 6 months. Symptoms include ulcerative cutaneous lesions (cutaneous leishmaniasis or CL), lesions in the mucosal areas of the mouth and/or nose (mucocutaneous leishmaniasis or MCL), and internal pathological manifestations resulting in fever, swollen lymph glands, anemia, enlargement of the liver and spleen, and progressive emaciation and weakness (visceral leishmaniasis or VL).

CL (Baghdad boil or Oriental sore) caused by infection with *Le. tropica* typically appears as a nonhealing ulcer and is often referred to as anthroponotic cutaneous leishmaniasis (ACL). The lesion usually develops within weeks or months of an infected sand fly bite and slowly evolves from a papule to a nodule to an ulcer. Cutaneous lesions may resolve quickly (2 to 3 months) without treatment or they may become chronic (lasting months to years) and will seldom heal without treatment. Scarring is associated with healing. In endemic areas, such scars are common among rural and urban populations. Life-long immunity to the infecting *Leishmania* species normally results.

CL caused by *Le. major* is often referred to as zoonotic cutaneous leishmaniasis (ZCL) and typically appears as 1 or more wet-looking non-healing ulcers. The lesion(s) usually develop within weeks after sand fly bites and quickly evolve from papules to open wet sores with raised, reddened edges. Lesions normally heal spontaneously and provide lifetime immunity against that species of *Leishmania*. Scars associated with healing are often evident in rural populations of South Central Asia.

VL (Kala-azar, Dum Dum fever), caused by *Le. infantum*, is a severe form of leishmaniasis, with as much as 95% mortality in untreated cases. It is a chronic disease that, without treatment, is marked by fever (2 daily peaks), weakness and, as the parasites invade internal organs, weight loss coupled with enlargement of spleen and liver that may resemble severe malnutrition. It should be noted that cutaneous lesions may also be seen in human visceral leishmaniasis cases, but the chronic visceralizing nature of the disease is the main concern. In South Central Asia, VL is usually a disease of young children and the elderly. The increasing incidence of AIDS has made many people more susceptible to VL and the complications of this and other infectious and vector-borne diseases. Viscerotropic *Le. tropica* has also been reported and was described in veterans of the Persian Gulf War, and several viscerotropic cases of *Le. tropica* have been reported from India and Pakistan. The incubation period for VL is usually 4 to 6 months but may be as short as 10 days or as long as 2 years. By the time the disease is diagnosed, patients have usually forgotten any contact with sand flies. Epidemics of VL often follow conditions of severe drought, famine or disruption of native populations by wars that produce large numbers of refugees. In Sudan, between the years 1991 and 1996, there were reports of 10,000 treated cases and an estimated 100,000 deaths from untreated cases of VL due to the shortage of health services.

Military Impact and Historical Perspective. Leishmaniasis is a persistent health threat to U.S. military personnel because troops deploy or conduct military exercises in locations where the disease is endemic. The potential for this disease to compromise mission objectives is significant. Soldiers exposed to sand fly bites while deployed to South Central Asia are highly susceptible to infection with *Leishmania tropica*. Immunity among US military personnel is essentially nonexistent, and recovery from CL does not confer immunity to VL. Most of the cases of visceral leishmaniasis (34 of 49) during World War II were acquired in India, in or near Calcutta. Accurate information is not available for incidence of cutaneous

leishmaniasis, since most cases were treated as outpatients. In the Karum River Valley of Iraq, US forces suffered 630 cases of VL in a three-month period during WWII. From 1990 to 91, 12 cases of VL due to *Le. tropica* were diagnosed when 697,000 allied soldiers were deployed to the Arabian Peninsula during Operations Desert Shield and Storm. Even though no fatalities were associated with leishmaniasis in this deployment, new lessons were learned that could affect future military deployments. Before the Persian Gulf War, eastern Saudi Arabia was not known to be endemic for visceral leishmaniasis, and *Le. tropica* was not convincingly shown to produce visceral disease. More important, the potential for leishmaniasis to cause post-deployment diagnostic problems and threaten blood supplies had not been anticipated. Returnees from the Persian Gulf War were barred from donating blood for up to 2 years, severely impacting blood supplies. Infection with *Leishmania* was even suspected as one of the causative agents of Persian Gulf War syndrome, but scientific evidence for this association was never established.

Diagnosis of leishmaniasis is difficult at best, and providing proper care for service members who may have been exposed or infected is a long, costly and complex process. Treatment usually requires 20 or more days and consists of injections with pentavalent antimony (Pentostam). Because this drug is not registered for use in the US, it must be administered under an experimental protocol at an approved medical treatment facility. Estimated leishmaniasis-related costs can exceed US $17,000 per patient, with an average of 92 lost duty days per patient. Other important but less quantifiable costs include loss to the unit, personal distress, and delay of career progression.

Disease Distribution. Anthroponotic (human-to-human) transmission of CL due to *Le. tropica* occurs in urban centers and rural highland villages of Afghanistan, Pakistan and India. The disease is widespread in areas with a temperate climate (Indian subcontinent), as well as those with an arid, cold climate (Transcaucasian region, Afghanistan, and Pakistan). Zoonotic (rodent to human) CL transmission due to *Le. major* occurs countrywide in Pakistan, in small villages or rural areas of Afghanistan and India, and in dry jungle areas of Sri Lanka. It is focally endemic and has been described as one of the major public health problems in Afghanistan, where the reservoir is the great gerbil, *Rhombomys opimus*. The endemic areas of CL appear in Figure 12.

Figure 12. Distribution of Cutaneous Leishmaniasis in South Central Asia.

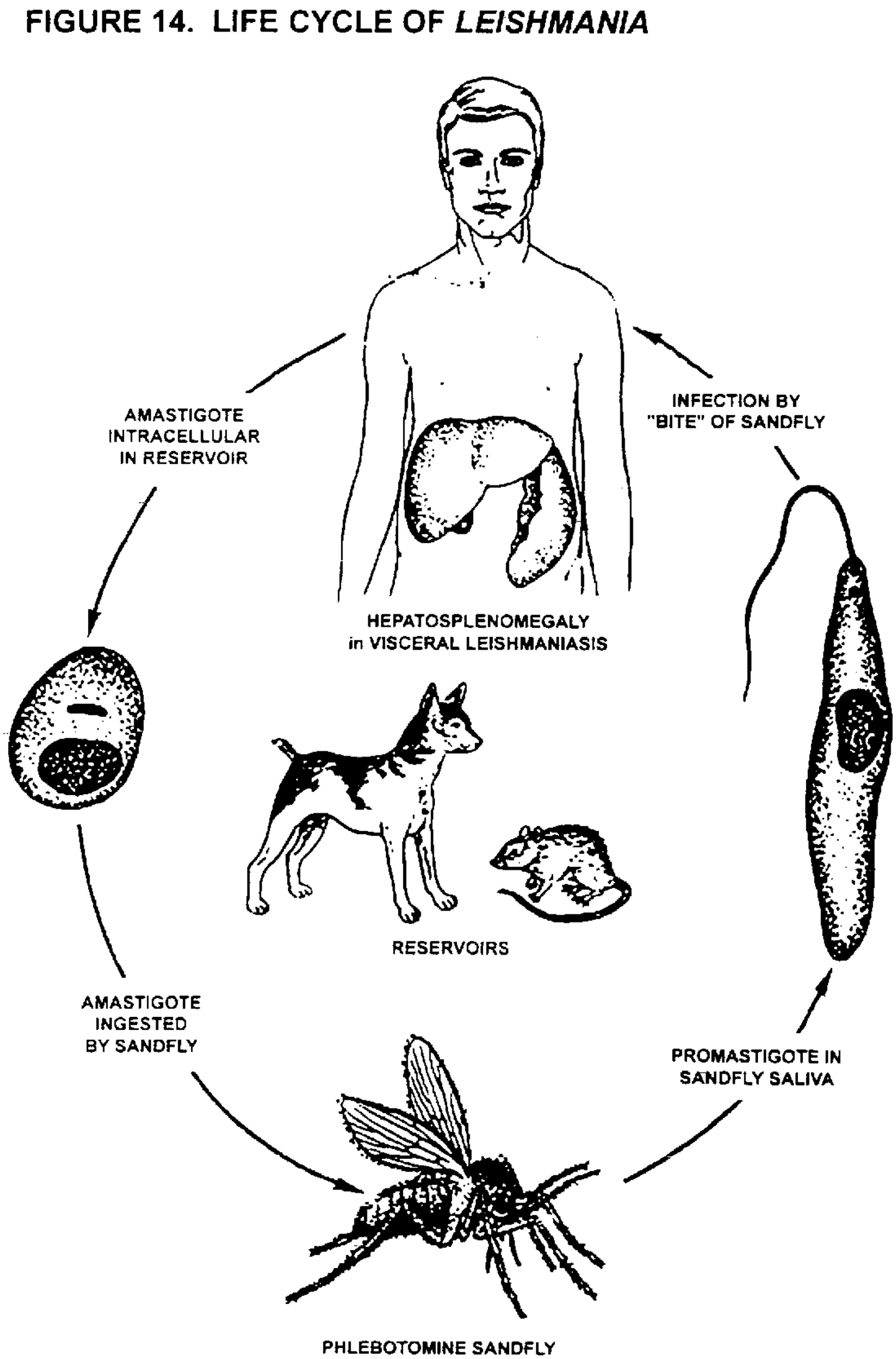
FIG. 12. DISTRIBUTION OF CUTANEOUS LEISHMANIASIS IN SOUTH CENTRAL ASIA (DARK SHADING).

VL due to *Le. infantum* occurs in the Mediterranean Basin countries of North Africa, the Middle East, and southern Europe, and in East Africa, South Central Asia, and China. It is replaced by *Le. donovani* in India, Bangladesh, Nepal, Pakistan and Afghanistan. *Leishmania donovani* strains resistant to the standard therapeutic agents sodium stibogluconate and pentamidine have been reported in India. VL caused by *Le. infantum* has occasionally been reported from Pakistan and Pakistan-administered Azad Kashmir and Northern areas. VL caused by *Le. donovani* is focally endemic in Afghanistan and the southeastern parts of Nepal. It is highly endemic in parts of India and Bangladesh, where transmission is year-round, though risk is elevated during the peak of sand fly activity from April through October. *Phlebotomus argentipes*, normally zoophilic on cattle, is strongly attracted to man where man is the suspected reservoir of VL caused by *Le. donovani* in South Central Asia. In many areas of the Indian Subcontinent, the incidence of leishmaniasis has increased dramatically following a reduction in the use of residual insecticides inside human habitations to control malaria. Endemic areas of VL appear in Figure 13.

Figure 13. Distribution of Visceral Leishmaniasis in South Central Asia.

Transmission Cycles. In urban areas humans may serve as reservoirs of *Le. tropica*. In rural areas, non-human hosts of *Le. tropica* may include wild and domestic rodents living in close proximity to humans. Sand fly vectors inhabit the burrow systems of domestic and wild rodents, moles, hedgehogs and jerboas, and acquire infections while feeding on these reservoir hosts. Amastigotes (the mammalian form of the *Leishmania* parasite) ingested with the bloodmeal transform to flagellated promastigotes within the gut of the female fly. In addition to a bloodmeal, the female consumes sugar in the nectar of nearby plants. These sugars help maintain *Leishmania* infections in the flies. Promastigotes multiply within the bloodmeal in the gut of the sand fly and undergo development to infective metacyclic promastigotes. By the time the bloodmeal is digested and the fly is ready to lay its eggs, infective metacyclic promastigotes are ready to be transmitted to the next vertebrate host when the sand fly feeds again (Figure 14).

Figure 14. Life Cycle of Leishmania.



In human hosts, infective-stage promastigotes (metacyclics) are engulfed by white blood cells or macrophages, in which they transform to amastigotes. Amastigotes proliferate in the macrophage until it

ruptures and new macrophages are invaded. At the skin surface, the tiny bite site becomes a small red papule that enlarges and ulcerates, with a raised edge of red inflamed skin. This inflamed area is where macrophages continue to engulf parasites, resulting in additional parasite multiplication. The ulcerated sores may become painful, last for months and, in uncomplicated CL caused by *Le. tropica*, eventually heal to form the characteristic scars seen on large numbers of people in some endemic areas of South Central Asia.

The incriminated vectors of *Le. tropica* in South Central Asia are *P. sergenti* and *P. caucasicus*. This form of CL ("dry" sore) is most common in densely populated urban centers and is considered to have a human-sand fly-human (anthroponotic cutaneous leishmaniasis, ACL) transmission cycle, with no recognized sylvatic reservoir. In recent years, ACL due to *Le. tropica* has been reported in rural highland villages of northern Pakistan and southern Afghanistan. In these foci, the disease is thought to have a zoonotic transmission cycle in which *P. kazeruni* and *P. mongolensis* maintain the parasite within a wild rodent reservoir population, although a sylvatic reservoir has not been confirmed.

The incriminated and proven vectors of *Le. major* (Zoonotic Cutaneous Leishmaniasis, ZCL) in South Central Asia are *P. alexandri*, *P. caucasicus*, *P. papatasi* and *P. salehi*. The proven vector of this "Wet" sore or "Rural" form of CL is *P. papatasi*. People who sleep outside with little clothing during warm weather in small villages and remote unpopulated areas often receive hundreds of bites from this species. Sand fly species suspected of circulating the parasite among rodent reservoirs are *P. kazeruni* and *P. mongolensis*.

The cycle of development of parasites causing VL is essentially the same as described for CL. The differences when dealing with VL caused by *Le. donovani* are the species of sand fly vectoring the disease (mainly *P. argentipes*, with *P. caucasicus*, *P. chinensis*, *P. major* and *P. longiductus* in some areas) and the disease reservoirs. In South Central Asia, VL is caused mainly by infection with *Leishmania donovani* and is thought to be an anthroponotic disease spread by the sand flies from man to man. Incidental infections in dogs are common where dogs live in close association with their owners. As mentioned earlier, a less virulent, viscerotropic form of *Le. tropica* has also been reported from the Middle East, India and Pakistan.

Vector Ecology Profiles. The incriminated vectors of *Le. tropica* (ACL) in South Central Asia are *P. sergenti* and *P. caucasicus*. Proven vectors of *Le. donovani* (VL) are *P. alexandrei* and *P. argentipes*. Suspected vectors of *Le. donovani* (VL) are *P. caucasicus*, *P. kandelakii*, *P. longiductus*, and *P. mongolensis*. Suspected vectors of *Le. infantum* (VL) in western Central Asia are *P. caucasicus*, *P. chinensis*, *P. kandelakii s.l.*, *P. longiductus* and *P. major.* The proven vector of *Le. major* (ZCL) in South Central Asia is *P. papatasi*. Incriminated vectors of *Le. major* (ZCL) are *P. alexandrei*, *P. caucasicus*, *P. salehi* and *P. sergenti*.

Adult sand flies rest during the daytime in dark, humid, protected areas, such as rodent burrows, rock crevices and caves. The preparation of military bunkered ground positions in desert areas provides additional protected daytime resting sites for phlebotomine sand flies. In urban areas, sand fly adults often rest in dark, cool, humid areas of human habitations and animal structures. Abandoned structures and their vegetative overgrowth often become attractive wild or domestic rodent habitats and foci of rural CL.

Nectar is important as a sugar source for both male and female sand flies, and sugars are required for development of parasite infections. After a bloodmeal, eggs are deposited in dark, humid, secluded areas. They develop into minute caterpillar-like larvae that feed on mold spores and organic debris. The larvae go through 4 instars and then pupate near larval feeding sites. Development from egg to adult requires 30 to 45 days, depending on feeding conditions and environmental temperatures. Phlebotomine sand fly eggs, larvae and pupae have seldom been found in nature, although exhaustive studies and searches have been made. The adult female has been observed to spread eggs around rather than oviposit in a single site. The larvae are widely distributed in the environment but are probably below the ground surface in termite mounds, rodent burrows, caves, or cracks and crevices in the soil where temperature, humidity and mold growth provide ideal conditions for larval development.

The dusk-to-dawn movement of adults is characterized by short, hopping flights just above the ground surface to avoid wind. Adult sand flies generally do not travel great distances, and most flights are believed to be less than 100 m. Sand fly habitats in the region range in altitude from desert areas below sea level to 3,500 m in the mountains. In temperate climates, adult sand flies are most abundant and active in the warmer months of April through October, especially after rains. However, species of sand flies such as *P. sergenti* are tolerant of cold winters and, along with *P. chinensis*, are found in the very mountainous (up to 3,500 m) northerly parts of India, Pakistan, Afghanistan and Nepal, where they will bite man in cool conditions. Both *P. sergenti* and *P. papatasi* will bite humans either indoors or outdoors and are troublesome pest species.

Female sand flies are quiet "stealth biters," from which comes the name "*papataci*" (silent gorger), and their bites may go unnoticed by military personnel. Sand flies may also bite during the daytime if disturbed in their secluded resting sites. Areas with some vegetation and cliffs, rock outcroppings, or other geologic formations that allow for suitable hiding places and daytime resting sites are important habitats. Exact information on reservoirs and vectors will require more extensive study in many countries of the region. Vast areas of these countries remain unsurveyed for sand fly vectors and disease. When searches are made, sand fly vectors are often found in areas where they were previously unknown.

Vector Surveillance and Suppression. See sand fly fever.

B. Schistosomiasis.

This disease, also known as Bilharziasis and snail fever, is caused by trematodes in the genus *Schistosoma* that live in the veins of humans and other vertebrates. Eggs from adult worms produce minute granulomata and scars in the organs where they lodge. Symptoms are related to the number and location of the eggs. The WHO considers 5 species of schistosomes significant in terms of human disease. *Schistosoma mansoni*, *S. japonicum*, *S. mekongi* and *S. intercalatum* give rise to primarily hepatic and intestinal symptoms. Infection with *S. haematobium* usually produces urinary manifestations. The most severe pathological effects are the complications that result from chronic infection. Symptoms of acute disease appear 2 to 8 weeks after initial infection, depending on the parasite species, and can be intense, especially in nonimmune hosts. Clinical manifestations include fever, headache, diarrhea, nausea and vomiting. Blood is usually present in the urine of well-established *S. haematobium* cases. The acute stage of schistosomiasis is usually more severe in the Asian forms *S. japonicum* and *S. mekongi* than in *S. mansoni*, *S. intercalatum*, or *S. haematobium*.

Military Impact and Historical Perspective. The first documented cases of schistosomiasis in US military personnel occurred in 1913 among sailors assigned to the Yangtze Patrol in China. Significant portions of the crews on some patrol boats were incapacitated. During World War I, American forces were not deployed in areas endemic for schistosomiasis. However, infection was prevalent among Allied Forces engaged in Mesopotamia and various parts of Africa. During World War II, the US Army hospitalized 2,088 patients with schistosomiasis. More importantly, an average of 159 days were lost per admission, almost half a year per case. Over 1,500 cases of acute infection due to *S. japonicum* were reported in US troops during the reinvasion of Leyte in the Philippines. Allied and Axis troops deployed in the North African and Middle East campaigns experienced high rates of infection. During the early 1950s, troops of the People's Republic of China were training along the Yangtze River for an amphibious landing on Taiwan. However, the invasion had to be cancelled when 30,000 to 50,000 cases of acute schistosomiasis, 10 to 15% of the invasion force, occurred. By the time the Chinese army recovered, the US had established the Taiwan Defense Command and had begun routine patrols of the Taiwan Strait. Schistosomiasis was rare among US military personnel during the Vietnam War.

Disease Distribution. Over 200 million persons are infected with schistosomiasis worldwide, causing serious acute and chronic morbidity. The absence of appropriate snail intermediate hosts in the Indian Subcontinent has led to the belief that human schistosomiasis cannot become established in the region. A focus of urinary schistosomiasis was identified in Madras State, India, in 1967 but was eradicated by 1973. An active focus of *S. haematobium* was identified in Gimvi village, Ratnagiri District, Maharashtra State, in 1952 and was confirmed as still active in the early 1980s. Although this focus may be limited or

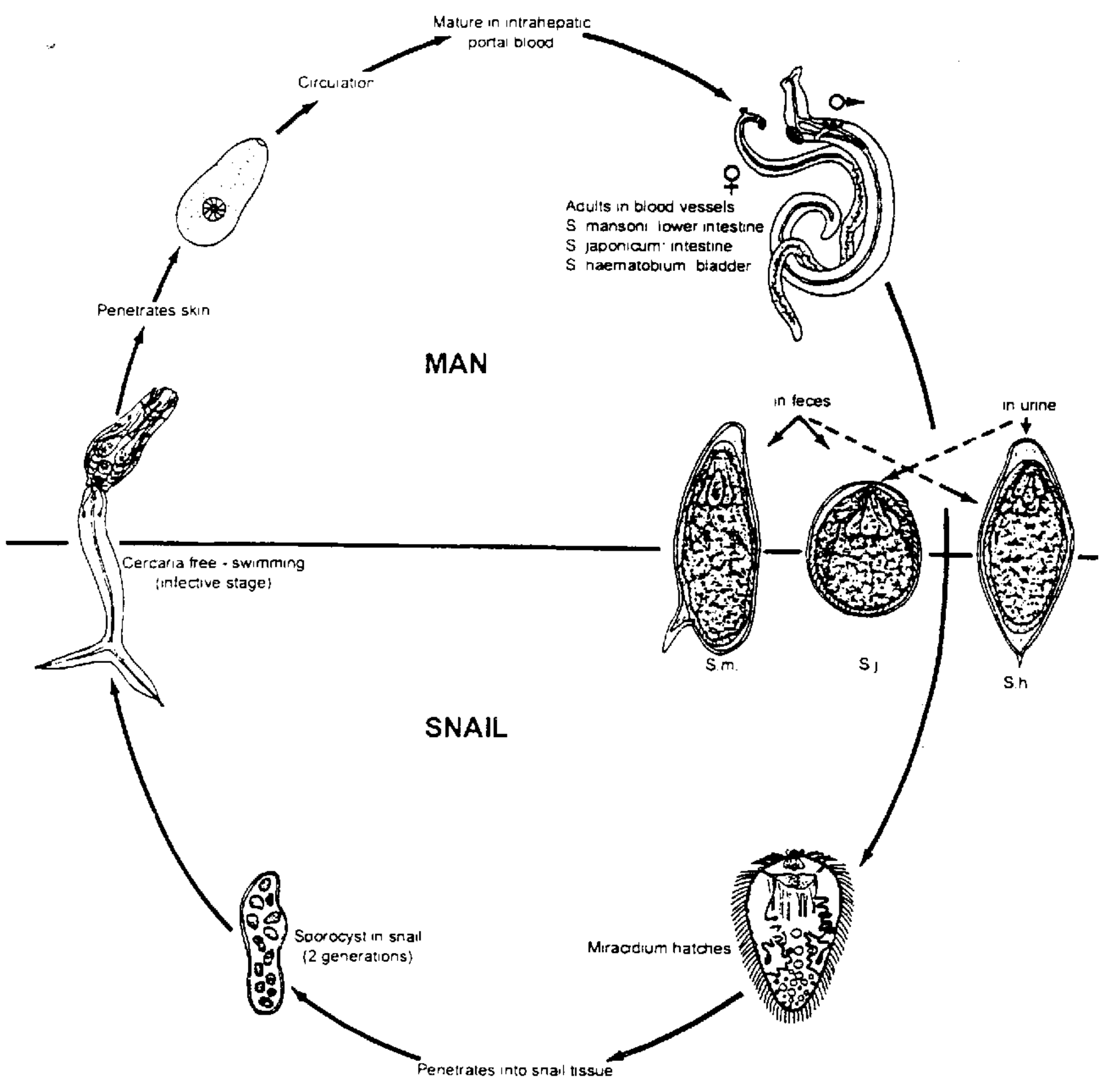
inactive, an enormous irrigation development on the Narmada River has the potential to spread any schistosomes in the area. The Narmada Valey Development Project is one of the largest schemes of its kind in the world and entails the construction of 30 major dams, 135 medium dams and 3,000 minor dams on the Narmada River and its tributaries. The possibility of additional foci of human schistosomiasis elsewhere exists. In the late 1980s, isolated cases of schistosomiasis were reported from Dokur, Andhra Pradesh, and from Burdwan District, West Bengal. The movement of Indian troops and Indian laborers to various African and Asian countries where schistosomiasis is endemic increases the threat of introducing this disease to the Indian Subcontinent. A serological study in Dhanusha District in southern Nepal in July 1996 and May 1997 indicated that *S. mansoni* probably exists in the area. This was the first report of *S. mansoni* in Nepal.

There are at least 8 species of animal schistosomes circulating among animal reservoirs in South Central Asia. Although these cannot cause disease in humans, exposure to their cercariae can produce a temporary, and sometimes severe, dermatitis. Rice field dermatitis is an occupational health problem this region. Rash and itching sensations occur within hours of exposure.

Transmission Cycle(s). The life cycles of the various schistosomes infecting man are similar. A generalized life cycle appears in Figure 15. Humans are infected when they are exposed to cercariae in infested fresh water. A single infected snail intermediate host may release 500 to 2,000 cercariae daily. Cercariae are infective for about 12 hours after being released from the snail. After cercariae penetrate the skin and enter the blood or lymph vessels, they are carried to blood vessels of the lungs before migrating to the liver, where they develop into mature adult male and female worms. They mate in the liver and migrate as pairs to veins of the abdominal cavity, usually the superior mesenteric veins in the case of intestinal forms (*S. mansoni*, *S. mekongi*, *S. intercalatum* and *S. japonicum*) or the venous plexus of the urinary bladder in the case of *S. haematobium*. Four to 6 weeks after initial penetration of the skin, adult females begin laying eggs. Female worms can deposit from 300 to 2,500 eggs per day. Adult worms live 3 to 7 years, but life spans of 30 years have been reported. Only about 50% of the eggs produced reach the bladder or intestine, where they are excreted in the urine and feces. The rest become lodged in the liver and other organs. The immunological reaction to the eggs is the primary cause of both acute and chronic clinical symptoms. The degree of chronic disease is directly related to the number of eggs deposited in the tissues. After excretion in urine or feces, a schistosome egg hatches in fresh water, releasing a single miracidium that infects an appropriate species of snail. The miracidium can survive as an infective free-living entity for less than a day. Miracidia undergo a complicated, asexual cycle of development and multiplication in the snail, but after 30 to 60 days each successful miracidium gives rise to several hundred infective cercariae. Humans are the principal hosts for both *S. mansoni* and *S. haematobium*, although natural infections of *S. mansoni* have been found in rodents and other mammals in Egypt.

Figure 15. Life Cycle of Schistosomes.

Figure 15. Life Cycle of Schistosomes



Vector Ecology Profiles.

General Bionomics.
Vector snails are focally distributed in rural and urban areas, associated with slow- moving streams, irrigation canals, oases, cisterns, and aqueducts. Expansion of the number of irrigation projects throughout South Central Asia has increased the habitats for snails. Concrete-lined, covered canals are usually poor habitats, while soil-lined canals that allow reeds or other marshy vegetation to grow provide excellent snail habitats. Tidal areas are not suitable habitats for snail hosts. Snails survive dry seasons by burrowing beneath riverbeds or under moist stones. Snails may be transported by man and sometimes by birds. Self-fertilization is common among these hermaphroditic snail species, a characteristic that enhances the dispersal of snails, since only a single founder is necessary to establish a colony. The movement of

military equipment from a snail-infested area can export snails of significant medical and economic importance to other regions (Consult TIM 31).

Specific Bionomics.
Ferrissia tenuis was identified as the most likely snail intermediate host at the Gimvi focus. This is surprising since *S. haematobium* is only known to be transmitted by snails belonging to the genus *Bulinus*. *Ferrissia* spp. are widely distributed throughout the Indian Subcontinent. The life cycle of this small ancylid snail is poorly known, but it is found in small streams. Its peak abundance occurs from February to May. During the June to September monsoonal period, snail habitats may be dispersed by flooding.

Vector Surveillance and Suppression. The most important preventive measure in reducing the incidence of schistosomiasis is avoidance of fresh water with infective cercariae. Assume that all fresh water in endemic areas is infested unless proven otherwise. The absence of snails in an area does not preclude infection, since cercariae can be transported considerable distances by water currents. Combat commanders and troops must be instructed in the risk of infection and measures for schistosomiasis prevention. No topical repellent is currently available that provides long-term protection against cercarial penetration. Experimental studies have shown the insect repellent DEET to provide a significant level of protection; however, the beneficial effects of DEET last only a few minutes because of its rapid absorption through the skin or loss from the skin surface from washing. When DEET is experimentally incorporated into liposomes (LIPODEET), its activity is prolonged for more than 48 hrs after a single application. Commercial formulations that can be used to protect against cercarial penetration may be available in the near future. Cercariae penetrate the skin rapidly, so efforts to remove cercariae after exposure by applying alcohol or other disinfectants to the skin have limited value. Standard issue BDUs offer substantial protection against penetration, especially when trousers are tucked into boots. Rubber boots and gloves can provide additional protection for personnel whose duties require prolonged contact with water containing cercariae.

Cercarial emergence from snails is periodic, and the numbers found in natural waters vary with the time of day. Light stimulates cercarial release for *S. mansoni* and *S. haematobium*. Minimal numbers of cercariae are present early in the morning and at night. Restricting water contact during peak cercarial density may reduce risk of infection. Avoid water contact in mid to late morning except where *S. japonicum* is endemic and in the Caribbean where *S. mansoni* is endemic. In those areas, noctural rodents are the primary hosts and their peak activity is in the late afternoon and early evening. However, stepping on and crushing an infected snail will release thousands of cercariae.

Cercariae are killed by exposure for 30 minutes to concentrations of chlorine of 1 ppm. Treating water with iodine tablets is also effective. Heating water to 50^{o}C for 5 minutes or allowing it to stand for 72 hours will render it free of infective cercariae. Water purification filters and reverse osmosis are effective in removing cercariae.

Molluscicides may be applied area-wide or focally by preventive medicine teams to eliminate snails from aquatic areas likely to be used by military personnel. Consult TIM 23, A Concise Guide for the Detection, Prevention and Control of Schistosomiasis in the Uniformed Services, and TIM 24, Contingency Pest Management Pocket Guide, for molluscicide recommendations and application techniques. There is little evidence that snail intermediate hosts have developed resistance to commonly used molluscicides like niclosamide.

C. Filariasis.

Bancroftian filariasis is caused by the nematode *Wuchereria bancrofti*, which normally resides in the lymphatic systems of infected humans. Eight to 12 months after infection, adult female worms release thousands of microfilariae (prelarval filarial worms) into the circulatory system. Acute reaction to infection includes swelling of lymph nodes, fever and headache, and allergic reaction to metabolic products of filariae. However, many individuals are asymptomatic in the early stages of infection. Female nematodes continue to produce microfilariae over the next 15 to 18 years. Chronic filariasis develops slowly, with recurrent episodes of fever and inflammation of the lymph glands. Microfilariae can obstruct the lymphatic

system, causing the legs, breasts or scrotum to swell to grotesque proportions, a chronic condition known as elephantiasis. This occurs only after repeated infections. Nearly half of all infections are clinically asymptomatic, although they have microfilariae circulating in their blood and have hidden damage to their lymphatic and/or renal systems. The death of numerous microfilariae resulting from drug therapy may cause severe immune reactions.
Brugian filariasis is caused by the nematodes *Brugia malayi* and *B. timori*. Clinical manifestations are similar to those of Bancroftian filariasis, except that the recurrent acute attacks of filarial fever and inflamation of the lymph glands are more severe, and elephantitis is usually confined to the legs below the knees.

Military Impact and Historical Perspective. Microfilariae of *W. bancrofti* were first discovered in the blood of a patient in Brazil in 1866. This was the first discovery of a pathogen transmitted by insects. Over 70 million people worldwide are estimated to be infected with *W. bancrofti*, resulting in serious economic costs to developing countries. The long incubation period and requirement for repeated infections before chronic clinical symptoms appear render Bancroftian filariasis of little medical significance to military operations. However, military personnel moving into an endemic area from one that is free from filariasis may develop acute symptoms such as swelling of the lymph glands, headache and fever months before larvae become mature. From 1942 to 1944, American military forces in the Samoan-Ellice-Wallis Islands rapidly developed swollen lymph glands and swollen extremities following repeated exposure to infected mosquitoes. Acute filariasis is the primary military concern, because its symptoms develop fairly rapidly and may be severe enough to cause removal of troops from their duties. Clinical manifestations of filariasis often occur with no demonstrable circulating microfilariae (occult filariasis). Of several thousand cases involving American military personnel during World War II, microfilariae were found in only 10 to 15 patients. In addition, the sight of people with grotesque deformities caused by chronic infection can have an adverse psychological impact. Medical personnel should be aware that troops with brief exposure to infection are often not diagnosed until after they return from deployments.

Disease Distribution. *Wuchereria bancrofti* occurs in most tropical and some subtropical regions, including Latin America, Africa, Asia and the Pacific islands. Mass migrations of infected humans are usually required to introduce the disease to new areas. The nocturnally periodic form of *B. malayi* occurs in rural populations living in open rice growing areas or near open swampy areas in Asia, from India to Japan. The subperiodic form of *B. malayi* is associated with swampy forests of Malaysia, Indonesia and the Philippines. Infections with *B. timori* occur on Timor and other southeastern islands of Indonesia.

It is estimated that 1 billion people are at risk of acquiring infection. Over 120 million people in at least 80 countries are currently infected with some form of filariasis, and 40 million of these are seriously incapacitated and disfigured by the disease. At least one-third of the people infected with the disease live in India. Ninety percent of infections worldwide are caused by *W. bancrofti*, and most of the remainder by *B. malayi*. Primary endemic areas of filariasis appear in Figure 16.

Figure 16. Distribution of Filariais in South Central Asia.

Bangladesh: Bancroftian filariasis may infect as much as 10% of the population. Prevalence of infection is highest in the northern and central districts.

India: Bancroftian filariasis is endemic countrywide, with the highest prevalence rates in Uttar Pradesh and Bihar States. Incidence has increased over the last 2 decades as a result of uncontrolled urbanization and the proliferation of breeding sites for its primary vector, *Cx. pipiens.* However, incidence of the disease has been increasing rapidly in rural areas as well. Malayan filariasis is most prevalent in the southern states of Andhra Pradesh, Orissa, Tamil Nadu and, especially, Kerala. It is also focally endemic in the states of Assam, Madhya Pradesh and West Bengal. Risk of transmission is usually higher during and after the rainy season.

Pakistan: Both Bancroftian and Malayan filariasis are endemic at low levels. The former is prevalent primarily in the southern Indus delta. Since 1947, large numbers of people have moved from Bangladesh

to settle in Pakistan. Many of these people came from areas where filariasis was endemic. A survey in 1980 found 9% of expatriates were infected with *W. bancrofti*.

Sri Lanka: Bancroftian filariasis is endemic in urban and rural areas within a western coastal belt extending from Kataragama in Southern Province to Puttalam in North Western Province. A few foci exist outside this area where populations of the vector *Cx. pipiens quinquefasciatus* are high.

Transmission Cycle(s). Microfilariae circulating in human blood are ingested by mosquitoes and undergo several days of development before the vector can transmit infective stages of the nematode. Infective parasites enter the bloodstream directly during a mosquito bite. A few nematode larvae are deposited on the skin and can enter the host through skin abrasions. In humans, larvae undergo development to adults that produce microfilariae for many years. Over most of the geographic range of this disease, including South Central Asia, *W. bancrofti* microfilariae exhibit pronounced nocturnal periodicity and consequently are ingested by night-biting mosquitoes. Peak abundance of microfilariae in the blood occurs between 2300 and 0300 hours. *Culex pipiens quinquefasciatus* is the most common urban vector. In rural areas, transmission is mainly by *Anopheles* spp. and *Culex* spp. There are no known animal reservoirs of Bancroftian filariasis.

There are no significant animal reservoirs for nocturnally periodic forms of *B. malayi* or *B. timori*. The subperiodic form of *B. malayi* infects humans, monkeys, (especially leaf monkeys, *Presbytis* spp.), wild and domestic cats, and pangolins (scaly anteaters). The zoonotic and epidemic life cycles of subperiodic *B. malayi* usually do not overlap. In Brugian filariasis, *Mansonia* spp. serve as the major vectors, but in some areas anopheline mosquitoes are responsible for transmitting the infection.

Vector Ecology Profiles. Vectors of filariasis are widespread in the region and include *Culex pipiens quinquefasciatus*, *Mansonia annulifera*, *Ma. indiana*, and *Ma. uniformis*. *Culex pipiens quinquefasciatus* is the primary vector of *Wuchereria bancrofti*, while *Ma. annulifera* is a secondary vector. *Mansonia annulifera*, *Ma. indiana*, and *Ma. uniformis* are vectors of *Brugia malayi*. *Anopheles stephensi* is a possible secondary vector of *B. malayi*.

Culex pipiens quinquefasciatus is the primary vector of *Wuchereria bancrofti* because it is highly anthropophilic, and its peak feeding activity, from 2200 to 0300 hours, coincides with the nocturnal periodicity of the microfilariae in human blood. *Culex pipiens quinquefasciatus* is widespread and abundant throughout the region. Adults usually prefer to feed on birds but readily feed on humans and large animals like cattle and goats. They feed early in the evening, usually within 2 hours of sunset, although feeding may continue throughout the night. Adults are strong fliers and will travel 3 to 5 km from breeding sites to find a blood meal. This species is an annoying biter and produces a high-pitched buzzing sound that can easily be heard. *Culex pipiens quinquefasciatus* feeds and rests indoors or outdoors. Before and after feeding indoors, they rest behind or under furniture and draperies, or in closets. Adults are more endophilic in the late fall, between November and December. Three or 4 days after a bloodmeal, *Cx. p. quinquefasciatus* deposits egg rafts containing 75 to 200 eggs on the water surface. Common oviposition sites include cisterns, water troughs, irrigation spillovers, wastewater lagoons, and swamps. Eggs hatch 2 to 4 days after deposition. Larvae of *Cx. p. quinquefasciatus* generally prefer ground pools with high concentrations of organic matter or swamps with emergent vegetation. Polluted water from septic systems is ideal for larvae of this species. Slums with poor sanitation and urban construction sites have proliferated in many areas and provide abundant breeding sites for *Cx. p. quinquefasciatus*. Larval development requires 7 to 9 days at a temperature range of 25 to 30°C. At lower temperatures, larval stages may require 15 to 20 days. The pupal stage lasts about 2 days. Adults of C*x. p. quinquefasciatus* occur from May to October in most of South Central Asia, although they may be found practically year-round in southern parts of the region or where sewage effluent keeps breeding sites warm. Adults usually display 2 population peaks, 1 from April to May and another from October to December.

The *Mansonia* species are good vectors of *Brugia* parasites because their peak biting activity nearly coincides with the nocturnal abundance of microfilariae in human blood. The bionomics of *Anopheles stephensi* is discussed in the section on malaria.

Mansonia annulifera is a highly anthropophilic species that is widespread in India, Nepal, and Bangladesh. In northeast India, this species is most abundant in the monsoon months of August to October, with a population peak in September. It is a strong flier (> 5 km) that feeds primarily indoors. It is a persistent biter and frequently occurs in large numbers. It feeds on man in strong preference to cattle and other animals. Its feeding activity increases steadily as the night progresses, displaying 2 peaks, one at 2400 to 0100 hours, and the other at 0400 to 0500 hours. Eggs are laid in sticky compact masses, often arranged as a rosette, and are glued to the undersurfaces of floating vegetation. Typical habitats include more or less permanent bodies of water with floating or submerged vegetation, primarily grasses, such as swamps, ponds, and fallow or active rice fields. *Mansonia* larvae are easily recognized: they have specialized pointed air siphons adapted for piercing aquatic plants to obtain air. Pupae also obtain oxygen from plant tissues by inserting their modified respiratory trumpets into plant stems.

Mansonia uniformis is not as common or as anthropophilic as *Ma. annulifera*. It is a persistent biter but is more zoophilic (feeding on cattle) than *Ma. annulifera*. Its peak biting activity is within the first hour after dusk, falling off steadily after that, with virtually no biting after 0300 hours. It is a strong flier, capable of traveling 5 km or more, and is primarily exophagic and exophilic.

Mansonia indiana is a relatively common species throughout the region, but mostly in lowland areas. Its period of greatest abundance in northeast India is from August to October, with a peak in September. This species tends to feed and rest indoors and outdoors in nearly equal numbers. It feeds on man and animals but prefers cattle to humans. *Mansonia indiana* has 2 peak biting periods during the night, one between 2000 to 2100 hours, and a second, smaller peak between 0300 and 0400 hours. Larvae of *Ma. uniformis* and *Ma. indiana* are found in the same habitats as *Ma. annulifera*.

Vector Surveillance and Suppression. Light traps are used to collect night-biting mosquitoes, but not all mosquito species are attracted to light. The addition of the attractant carbon dioxide to light traps increases the number of species collected. Traps using animals, or even humans, as bait are useful for determining feeding preferences of mosquitoes collected (use of humans as bait must be conducted under approved human-use protocols). Adults are often collected from indoor and outdoor resting sites using a mechanical aspirator and flashlight. Systematic larval sampling with a long-handled white dipper provides information on species composition and population dynamics, which is used when planning control measures.

Mosquitoes can be individually dissected and examined for filarial infection. Large numbers of mosquitoes can be processed more quickly by crushing them in a saline solution and removing filarial worms with a sieve. The parasites can then be concentrated by centrifugation. Careful identification is required so as not to confuse medically important species of filarial worms with those that chiefly infect nonhuman hosts.

Application of residual insecticides to the interior walls of buildings and sleeping quarters is an effective method of interrupting filarial transmission when local vectors feed and rest indoors. Nightly dispersal of ultra low volume (ULV) aerosols can reduce exophilic mosquito populations. Larvicides and biological control with larvivorous fish can reduce larval populations before adults have an opportunity to emerge and disperse. However, it is necessary to maintain vector density at low levels for prolonged periods to control filariasis. Hence chemotherapy of infected persons has been the chief tool to control the disease in endemic areas. Insecticides labeled for mosquito control are listed in TIM 24, Contingency Pest Management Pocket Guide. Chemical control may be difficult to achieve in some areas. After decades of insecticide use, many species of the *Cx. pipiens* complex are now resistant to insecticides (Appendix C. Pesticide Resistance in South Central Asia.) Sanitary improvements, such as filling and draining areas of impounded water to eliminate breeding habitats, should be used to the extent possible. Placing non-toxic expanded polystyrene beads (2 to 3 mm in diameter) into pit latrines and cess pits to completely cover the water surface with a 2 to 3 cm thick layer prevents *Culex* spp. from laying eggs in such places. A single application can persist for several years and give excellent control. *Mansonia* mosquitoes are usually controlled by removing or killing the aquatic weeds upon which the larvae and pupae depend for their oxygen requirements. If insecticides are used to control *Mansonia* larvae, granules or pellets are more suitable than liquid formulations because they can penetrate the vegetation layer and sink to the bottom of breeding places to release chemicals toxic to the larvae.

The proper use of repellents and other **personal protective measures** is thoroughly discussed in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance. The use of bednets impregnated with a synthetic pyrethroid, preferably permethrin, is an extremely effective method of protecting sleeping individuals from mosquito bites. The interior walls of tents can also be treated with permethrin. Buildings and sleeping quarters should be screened to prevent entry of mosquitoes and other blood-sucking insects.

VII. Other Diseases of Potential Military Significance.

A. Leptospirosis.

This disease is also known as Weil disease, Canicola fever, hemorrhagic jaundice, mud fever, and swineherd disease. The spirochete bacterium *Leptospira interrogans* is the causative agent of this zoonotic disease. More than 200 serovars of *L. interrogans* have been identified, and these have been classified into 23 serogroups based on serological relationships. Common clinical features are fever with sudden onset, headache, and severe muscle pain. Serious complications can occur. Infection of the kidneys and renal failure is the cause of death in most fatal cases. The severity of leptospirosis varies greatly and is determined to a large extent by the infecting strain and health of the individual. In some areas of enzootic leptospirosis, a majority of infections are mild or asymptomatic. The incubation period is 10 to 12 days after infection.

Disease Distribution. Leptospirosis is one of the most widespread zoonosis in the world. Distribution is worldwide in urban and rural areas of both developed and developing countries. Leptospirosis is regarded as focally enzootic throughout South Central Asia, and sporadic human cases are reported from most countries as a result of occupational exposure. The highest incidence of leptospiral antibodies is usually found in veterinarians and workers in animal husbandry. However, nearly 11% of military recruits from northern Pakistan undergoing training near Mardan in 1984 had a 4-fold rise in IgM antibody to leptospirosis. Several outbreaks of leptospirosis with high case fatality rates occurred in tribes of the Andaman and Nicobar Islands during the late 1980s. Seropositivity rates among tribes ranged from 14.8% to 53.5%. The seroprevalences of *Leptospira* antibodies in cattle, sheep, goats, rats and agricultural workers tested during 1995 in Punpozhil, a village in Tamilnadu State, India, were 61.5%, 56.3%, 75.0%, 52.9% and 72.5%, respectively. A previous study in 1993 on the prevalence of leptospirosis in Tamilnadu State detected leptospiral antibodies in 16 of 98 dogs (16%), 102 of 189 sheep (54%), 101 of 213 goats (47%), 20 of 57 buffalo (35%), 356 of 808 cattle (44%) and 229 of 833 humans (28%).

Transmission Cycle(s). *Leptospira* infects the kidneys and is transmitted in the urine of infected animals. Humans become infected through contact of skin or mucous membranes with contaminated water, moist soil or vegetation. *Leptospira* survives only in fresh water. Spirochetes are not shed in the saliva; therefore, animal bites are not a source of infection. Although infected humans shed *Leptospira* in urine, person-to-person transmission is rare. Infection may occasionally occur by ingestion of food contaminated with urine from infected rats. Infection from naturally infected meat or milk is low. Spirochetes disappear from whole milk within a few hours. Because of its prevalence in rodents and domestic animals, leptospirosis has usually been an occupational hazard to farmers, sewer workers, veterinarians, animal husbandry workers, slaughterhouse workers, and rice and sugarcane field workers. During a study in the early 1990s, nearly 33% of sanitation workers in Madras City had leptospiral antibodies.

Numerous wild and domestic animals act as reservoirs, including rodents, swine, cattle, sheep, goats, buffalo, camels, horses and even elephants. Nearly 33% of buffalo from an urban area (metropolitan Lahore) and 16% of buffalo from a rural area of Skeikhupura District in Pakistan's Punjab Province had antibodies to *L. interrogans* when tested in 1995. Cats and dogs are frequently infected but are probably insignificant as a source of infection to humans. Dogs are a good indicator of the distribution of different leptospiral serovars in the environment. Many small mammals are involved in the epidemiology of leptospirosis in South Central Asia. Leptospiral isolations were made from 89 of 500 *Rattus rattus* trapped in human dwellings in the Indian state of Karnataka during the late 1990s. Bandicoot rats, *Bandicota indica* and *B. bengalensis*, are important reservoirs in South Central Asia in addition to domestic rodents. Both species of bandicoots have become common commensals of humans. *Bandicota indica* is also

common in lowland rice fields. The incidence of leptospirosis has increased in areas where irrigation and year-round cultivation provide food and cover to host rodents. Close association of humans, animals, soil and water facilitates the spread of leptospirosis to humans. Leptospirosis is common in flood-prone districts of Bangladesh. Uveitis (inflamation of the vascular middle coat of the eye)is a rare complication of leptospirosis, though epidemics of uveitis among patients with leptospirosis have occurred in southern India.

Disease Prevention and Control. To prevent leptospirosis, control domestic rodents around living quarters and food storage and preparation areas. *Leptospira* are readily killed by detergents, desiccation, acidity, and temperatures above 60EC. Good sanitation reduces the risk of infection from commensal rodents. Troops should be educated about modes of transmission and instructed to avoid swimming or wading in potentially contaminated waters. Leptospirosis could be a problem following flooding of contaminated streams or rivers. Vaccines have been used effectively to protect workers in veterinary medicine, and immunization has also been used to protect against occupational exposure to specific serovars in Japan, China, Italy, Spain, France, and Israel. Short-term prophylaxis may be accomplished by administration of antibiotics. Doxycycline was effective in Panama in preventing leptospirosis in military personnel.

B. Hantaviral Disease.

This disease is known by many names including: epidemic hemorrhagic fever; Korean hemorrhagic fever; Nephropathia epidemica; hemorrhagic nephrosonephritis; hemorrhagic fever with renal syndrome (HFRS); hantavirus pulmonary syndrome (HPS). Hantaviruses are a closely related group of zoonotic viruses that infect rodents. The genus *Hantavirus*, family Bunyaviridae, comprises at least 14 viruses, including those that cause HFRS and HPS. Syndromes in humans vary in severity but are characterized by abrupt onset of fever, lower back pain, and varying degrees of hemorrhagic manifestations and renal or pulmonary involvement. Depending in part on which hantavirus is responsible for illness, HFRS can appear as a mild, moderate or severe disease. Severe illness is associated with Hantaan virus (HTN) and Seoul virus (SEO), primarily in Asia and the Balkans. The case fatality rate is variable but is about 5% in Asia and somewhat higher in the Balkans. Convalescence takes weeks to months. A less severe illness caused by Puumala virus (PUU) and referred to as nephropathia epidemica predominates in Europe. Dobrava-Belgrade (DOB) virus has caused severe HFRS cases in several countries surrounding Turkey, with mortality rates up to 20%. Tula virus (TUL) has been isolated throughout Europe and eastward to the Kirov region, Russia, but its role in human disease is unclear. HPS, caused by several hantaviruses, has been reported throughout North and South America.

Military Impact and Historical Perspective. Prior to World War II, Japanese and Soviet authors described HFRS along the Amur River in Manchuria. An epidemic of “trench nephritis” during World War I may have been due to hantaviral infection. Thousands of cases of this illness, considered an entirely new disease, were noted on both sides of the front. During World War II, more than 10,000 cases of a leptospirosis-like disease were recorded during the 1942 German campaign in Finnish Lapland. When the snow melted, great numbers of lemmings and field mice invaded German bunkers. In 1951, HFRS was recognized among United Nations troops in Korea and has been observed in both military personnel and civilians since then.

Hantaviral disease is an emerging medical threat to military forces operating in many areas of the world. Over 20 acute PUU infections were documented in US Army personnel during a 1990 field exercise in southern Germany. Several outbreaks of hantaviral disease occurred in 1995 as a result of the civil war in the states of the former Yugoslavia. Over 300 patients, most of them soldiers exposed in the field, were hospitalized in the Tuzla region (northeast Bosnia) with acute hantaviral disease. Outbreaks also occurred around Sarajevo and Zenica. Croatia reported over 200 cases from several localities. Over 100 cases were reported in northern Montenegro. Hantaviral infections also occurred during the fighting in Kosovo. Advanced diagnostic techniques have led to increasing recognition of new hantaviruses and hantaviral infections globally. New outbreaks with novel hantaviral strains are still being uncovered. The distribution of new and old strains of hantaviruses presents a complex and confusing epidemiological picture of this emerging disease, but the military threat is highly significant.

Disease Distribution. At least 200,000 cases of HFRS involving hospitalization are reported annually throughout the world. Hantaan virus claims 40,000 to 100,000 victims annually in China. South Korea has reported about 1,000 cases annually in recent years. SEO virus is associated with the commensal Norway rat and occurs in more urban areas. Most clinical cases are reported from the Balkans.

Little published information is available about the hantaviruses that may be circulating in South Central Asia. In India, antibodies have been found in humans for Puumala and Hantaan viruses, although there are few reported clinical cases. One positive human case for Hantaan virus was reported from the Andaman and Nicobar Islands during 1988. Thottapalayam, a novel hantavirus, has been isolated from *Suncus* shrews in India, but this virus has not been associated with human disease. No human cases have been reported from Nepal, but hantaviral antibodies have been found in hospital outpatients in Kathmandu. Seoul and Hantaan viruses are enzootic in Sri Lanka. Seoul virus has been isolated from rats in Colombo, and antibodies have been found in humans. A 1991 countrywide survey found that 27% of rural agricultural residents were seropositive for Hantaan virus. Additional epidemiological studies are needed to clarify the public health threat of hantaviral disease in South Central Asia.

Transmission Cycle(s). Virus is present in the urine, feces and saliva of persistently infected, asymptomatic rodents. Aerosol transmission to humans from rodent excreta is the most common mode of infection. Human-to-human transmission of HFRS is considered rare, although viruses have been isolated from the blood and urine of patients. Hantaviruses have caused laboratory-associated outbreaks of infection.

Each hantavirus appears to have a single predominant murid reservoir. HTN is commonly associated with the field mouse, *Apodemus agrarius*, in open field or unforested habitats. The red bank vole, *Clethrionomys glareolus*, inhabits woodland or forest-steppe environments and is a primary reservoir for PUU. DOB has been isolated from the yellow-necked field mouse, *Apodemus flavicollis*, in open field and unforested foothills. The Norway rat, *Rattus norvegicus*, is the reservoir for SEO worldwide. The common European vole, *Microtus arvalis*, appears to be the primary reservoir of TUL. Hantavirus infection is not pathogenic in its rodent reservoir and produces chronic and probably lifelong infection. Hantaviruses may be spread by infected rodents that infest ships, thereby reaching ports worldwide.

The risk of transmission is highest in warm months when rodent reservoir populations are abundant. Military personnel are exposed to infection when working, digging or sleeping in fields infested by infected rodents.

Disease Prevention and Control. Prevent rodent access to buildings. Store food in rodent-proof containers or buildings. Disinfect rodent-contaminated areas with dilute bleach or other antiviral agents. Do not sweep or vacuum rodent-contaminated areas; use a wet mop moistened with disinfectant. Eliminate wild rodent reservoirs before military encampments are established in fields. Do not disturb rodent droppings or sleep near rodent burrows. Military personnel should not handle or tame wild rodents. Rodents frequently urinate or bite when handled. Detailed information on surveillance and personal protective measures when working around potentially infected rodents can be found in TIM 40, Methods for Trapping and Sampling Small Mammals for Virologic Testing, and in TIM 41, Protection from Rodent-borne Diseases.

VIII. Noxious/Venomous Animals and Plants of Military Significance.

A. Arthropods.

Annoyance by biting and stinging arthropods can adversely affect troop morale. The salivary secretions and venoms of arthropods are complex mixtures of proteins and other substances that are allergenic. Reactions to arthropod bites and stings range from mild local irritation to systemic reactions causing considerable morbidity, including rare but life-threatening anaphylactic shock. Insect bites can be so severe and pervasive that they affect the operational readiness of troops in the field. Bites and their discomfort have been a major complaint by soldiers deployed in many regions of the world.

Entomophobia, the irrational fear of insects, and the related arachnophobia, fear of spiders, are two of the most common human phobias. The fear is usually not limited to obvious threats, such as scorpions The anxiety produced in a fearful individual by a potential encounter with an insect range from mild aversion to panic. The degree of negative response to encounters with insects or spiders is important in assessing the difference between common fear and true phobia. Common fear is a natural extension of human experience and is appropriate to situations that involve potential danger or require caution. Phobias, however, are characterized by persistent, high levels of anxiety in situations of little or no threat to the individual. Many individuals may express a fear of insects or spiders, but few are phobic to the extent that their ability to function in a normal daily routine is impaired by their fear. The term delusory parasitosis refers to a mental disorder in which an individual has an unwarranted belief that insects or mites are infesting his or her body or environment. This psychiatric condition is distinct from entomophobia or an exaggerated fear of real insects. Extreme entomophobia and delusory parasitosis require psychological treatment.

The following groups of noxious arthropods are those most likely to be encountered by military personnel operating in countries of South Central Asia:

1. Acari (ticks and mites).

Scabies is the most important disease caused by mite infestation, and infestations have been common during military conflict. During World War I, scabies infestations occurred at a rate of 20 per 1,000 soldiers per year among American forces in Europe. During World War II, nearly 100,000 cases were reported in American troops. Five percent of the residents of London became infested with scabies during the bombing of the city by the German Air Force. During the Falklands War of 1982, scabies became such a problem among Argentine troops that their fighting efficiency was significantly impaired.

Sarcoptes scabiei (family Sarcoptidae) is a parasitic mite that spends its entire life cycle in burrows in the skin of mammals. Mite infestations cause scabies in man and mange in other animals, including primates, horses, wild and domestic ruminants, pigs, camels, rabbits, dogs and other carnivores. Populations found on different host species differ physiologically more than morphologically and are referred to as forms (those on man, for instance, are *S. scabiei* form *hominis*). Some authors may refer to forms as varieties or even subspecies. All forms are considered to be the same species, *S. scabiei*. Mites from one host species do not establish themselves on another. Humans can become infested with scabies mites from horses or dogs, but such infestations are usually mild and disappear without treatment. *Sarcoptes* mites are common on domestic animals in South Central Asia, especially stray dogs, and troops should avoid contact with local animals.

Scabies mites principally burrow in the interdigital and elbow skin, but skin of the scrotum, breasts, knees and buttocks is also affected. The face and scalp are rarely involved. Scabies mites are very small, about 0.2 to 0.4 mm. Both sexes burrow in the horny layer of the skin, but only the female makes permanent burrows parallel to the skin surface. Burrowing may proceed up to 5 mm per day, and the burrow may extend over a cm in length. Mites feed on liquids oozing from dermal cells that have been chewed. Females lay 1 to 3 eggs per day in their tunnels and rarely leave their burrows. Eggs hatch into six-legged larvae that crawl out of the burrows onto the surface of the skin. Larvae burrow into the skin or a hair follicle and form a pocket in which they molt into the nymphal stage. Nymphs molt into adults, and the male burrows into the molting pocket in the skin and mates with the female. The female begins to burrow through the skin only after fertilization. Adult males can be found in short burrows or pockets in the skin or wandering around on the skin surface. The life cycle from egg to adult takes about 10 to 14 days. Adult females live about a month on humans but survive only a few days off the host. Clothing or bedding kept unused for about 5 days is usually free of mites.

Scabies is transmitted from person to person by close, prolonged personal contact. Transmission is common in dormitories, barracks and medical facilities. It is possible to get infested by sleeping in a bed formerly occupied by an infested person, but experimental work has indicated this rarely happens. Exposure for ten minutes at 50EC will kill mites. In newly infested persons, a period of 3 to 4 weeks usually elapses before sensitization to mites and mite excretions develops. Itching is not experienced

during this period, and infestations may progress extensively before being noticed. However, fewer than 20 mites are enough to produce intense itching, particularly at night. The burrows often become secondarily infected with bacteria causing pustules, eczema and impetigo. In infested persons, an extensive rash can cover areas where there are no mites, and a rash may persist for several weeks after all scabies mites have been killed. In immunocompromised individuals, who do not respond to infestation by itching and scratching, mites can reach very high populations and produce a scaly crusted skin known as Norwegian or crusted scabies. With a highly contagious condition like scabies it is important to treat all persons living in close association with an infested individual.

Sarcoptes scabiei is cosmopolitan and infestations are common in South Central Asia. Persons of all ages are affected, although in most developing countries infestation is highest in poor communities and in children. Infestation is more common in overcrowded areas with poor hygiene. A study in urban areas of Dhaka, Bangladesh, followed 766 children less than 6 years of age from October1984 to September 1985. During this period, 589 (77%) became infested with scabies. An epidemiological survey conducted during the early 1980s in the rural village of Baskripal Nagar, Alwar, India, examined 2,771 persons among 404 households. Prevalence rates were 13% by population and 31% by household. Significantly lower rates of infestation were found among the more prosperous households.

Scabies is not a reportable disease in most countries; thus, estimated rates of infestation are usually inaccurate. Scabies is usually only reported when large outbreaks occur. Increases in the incidence of scabies appear to occur in 15 to 20 year cycles that are related to fluctuating levels of immunity to *S. scabiei* in the human population.

Larvae of the mite family Trombiculidae are known variously as chiggers, harvest mites and scrub itch mites and are parasites of mammals and birds. Over 3,000 species have been described worldwide but only about 30 of these are known to attack humans. Larvae are very small, measuring about 0.25 mm long and are often called red bugs. Females lay eggs in damp soil. The eggs hatch into six-legged larvae that congregate near the tips of grass and fallen leaves and attach to passing animals that brush against the vegetation. Larvae cluster in the ears of rodents and around the eyes of birds. On humans they most often attach where the clothing is tight, around the waist or genitals. Chigger larvae do not burrow into the skin as commonly believed, nor do they feed primarily on blood. Larvae remain on the skin surface and use digestive fluids to form a feeding tube (stylostome) that enables them to feed on cellular material for several days. Fully fed larvae drop to the ground to continue their complex life cycle. In the nymphal and adult stages, they are believed to prey on the eggs and larvae of other arthropods. Feeding by chiggers can cause an intense itchy dermatitis leading to pustules and sometimes to secondary infection. Most temperate zone chiggers have one annual generation.

Tick paralysis is a potentially fatal but easily cured affliction of man and animals. It is almost exclusively associated with hard (ixodid) ticks and is caused by injection of neurotoxin(s) in tick saliva. The toxin, which may be different in different species, disrupts nerve synapses in the spinal cord and blocks the neuromuscular junctions. Worldwide, nearly 50 species of hard ticks have been associated with tick paralysis, although any ixodid tick may be capable of producing this syndrome. A tick must be attached to its host for 4 to 6 days before symptoms appear. This condition is characterized by an ascending, flaccid paralysis, usually beginning in the legs. Progressive paralysis can lead to respiratory failure and death. Diagnosis simply involves finding the embedded tick, usually at the base of the neck or in the scalp. After tick removal, symptoms resolve within hours or days. However, if paralysis is advanced, recovery can take several weeks. No drugs are available for treatment.

2. Araneae (spiders).

More than 35,000 species of spiders have been described worldwide. All spiders, with the exception of the family Uloboridae, are venomous and use their venom to immobilize or kill prey. Most spiders are harmless because their chelicerae cannot penetrate human skin, or they have venom of low toxicity to humans. Only about a dozen species have been responsible for severe systemic envenomization in humans, although as many as 500 species may be capable of inflicting significant bites. Those that can bite humans are rarely seen or recovered for identification, so physicians need to be able to recognize signs and

symptoms of common venomous spider bites in order to administer appropriate therapy. In South Central Asia the widow spiders, *Lactrodectus* spp. (family Theridiidae), and the sac spiders, *Chiracanthium* spp. (family Clubionidae), are responsible for significant local and systemic effects from envenomization.

The brown widow, *L. geometricus*, and the black widow, *L. mactans*, are widespread throughout the region. These are also referred to as hourglass, shoe button, or po-ko-moo spiders. Considerable variation in coloration and markings exists between species and between immatures and adults. Widow spiders are found in various habitats in the wild, especially in protected places, such as crawl spaces under buildings, holes in dirt embankments, piles of rocks, boards, bricks or firewood. Indoors, they prefer dark areas behind or underneath appliances, in deep closets and cabinets. They commonly infest outdoor privies, and preventive medicine personnel should routinely inspect these structures. Widow spiders spin a crude web and usually will not bite unless provoked.

Latrodectus spp. inject a potent neurotoxin when biting. The bite itself is mild and most patients don't remember being bitten. Significant envenomization results in severe systemic symptoms, including painful muscle spasms, a rigid board-like abdomen, and tightness in the chest. Mortality rates from untreated bites have been estimated at 1 to 5%. Most envenomizations respond quickly to sustained intravenous calcium gluconate. Antivenins are commercially available and very effective.

Sac spiders of the genus *Chiracanthium* have a cytolytic venom that produces cutaneous necrosis in victims, although the necrotizing lesions are usually not as severe as those produced by the bite of *Loxosceles* spp. Some species have neurotoxic components in their venom. Over 150 species of sac spiders have been recorded worldwide. The consequences of bites include severe local pain, fever, swelling and redness, with a small area of necrosis at the site of the bite.

3. Ceratopogonidae (biting midges, no-see-ums, punkies).

The Ceratopogonidae is a large family containing nearly 5,000 species. These extremely small flies can easily pass through window screens and standard mosquito netting, although most species feed outdoors. Their small size is responsible for the moniker "no-see-ums." Many species in this group attack and suck fluids from other insects. Most species that suck vertebrate blood belong to the genera *Culicoides* (1,000 species) or *Leptoconops* (about 80 species). In South Central Asia these insects do not transmit human diseases, but they do serve as vectors for several diseases of veterinary importance. Many species of Ceratopogonidae are widespread in the region, but little is known about their biology. Many South Central Asian species of *Culicoides* are zoophilic. *Leptoconops* are more likely to be a major nuisance to man. *Leptoconops spinosifrons* breeds around beaches and seriously interferes with tourism in India and Sri Lanka. Blood-sucking species predominately feed and rest outdoors, entering houses in much smaller numbers. Only females suck blood. *Leptoconops* are active during the day; *Culicoides* may be either diurnal or nocturnal. Diurnal species of both genera prefer early morning and late afternoon periods. Despite their small size, they often cause local reactions severe enough to render a military unit operationally ineffective. In sensitive people bites may blister, exude serum and itch for several days, or be complicated by secondary infections from scratching. Enormous numbers of these tiny flies often emerge from breeding sites, causing intolerable annoyance. Some species of *Culicoides* and *Leptoconops* are known to fly 2 to 3 km without the assistance of wind. Ceratopogonidae are troublesome mainly under calm conditions, and the number of flies declines rapidly with increasing wind speed.

Breeding habits vary widely from species to species. The larvae are primarily aquatic or semiaquatic, occurring in the sand or mud of fresh, salt, or brackish water habitats, notably salt marshes and mangrove swamps. Many species exploit specialized habitats such as tree holes, decaying vegetation, and cattle dung. Most species remain within 500 m of their breeding grounds. Ceratopogonidae are troublesome mainly under calm conditions, and the number of flies declines rapidly with increasing wind speed. In militarily secure areas, encampments should be located in the open, away from breeding sites, to avoid the nuisance caused by these insects.

Larvae are difficult to find, but adults are easily collected while biting and with light traps. Environmental management best controls larval stages, but this may be impractical in extensive or diffuse habitats.. Adult

control typically includes applying residual insecticides to fly harborages, treating screens and bednets with pyrethroids, and using repellents. Ultra low volume application of aerosols may produce temporary control, but sprayed areas are soon invaded by midges from unsprayed areas. Ceratopogonids have difficulty biting through clothing because of their short mouthparts, so even an untreated BDU can provide considerable protection.

4. Chilopoda (centipedes) and Diplopoda (millipedes).

Centipedes in tropical countries can attain considerable size. Members of the genus *Scolopendra* can be over 25 cm long and are capable of inflicting painful bites, with discomfort lasting 1 to 5 hours. Several species of this genus known to bite man occur in South Central Asia. *Scolopendra morsitans* is a widespread tropical species that is most often incriminated in human bites. Specimens up to 13 inches in length have been collected in the Andamans. Its bite produces excruciating local pain and burning. Smaller centipedes, such as *Otostigmus ceylonicus*, are occasionally reported as biting humans in the region. Centipedes bite using their first pair of trunk appendages (maxillipeds) which have have evolved into large, claw-like structures. Two puncture wounds at the site of attack characterize a centipede bite. Neurotoxic and hemolytic components of a centipede's venom normally produce only a localized reaction, but generalized symptoms such as vomiting, irregular pulse, dizziness and headache may occur. Most centipede bites are uncomplicated and self-limiting, but secondary infections can occur at the bite site. Centipede bites are rarely fatal to humans.

Centipedes are flattened in appearance and have 1 pair of legs per body segment. Large species may have over 100 pairs of legs. They are fast-moving, nocturnal predators of small arthropods. During the day, they hide under rocks, boards, bark, stones and leaf litter, but occasionally they find their way into homes, buildings, and tents. Centipedes are not aggressive and seldom bite unless molested. Most centipede bites occur when the victim is sleeping or when putting on clothes in which centipedes have hidden. Troops should be taught to inspect clothing and footwear when living in the field.

Millipedes are similar to centipedes except that they have two pairs of legs per body segment and are rounded or cylindrical instead of flattened. Millipedes are commonly found under stones, in soil and in leaf litter. They are nocturnal and most species feed on decaying organic matter. They are more abundant during the wet season. When disturbed they coil up into a tight spiral. Millipedes do not bite or sting, but some species secrete defensive body fluids containing quinones and cyanides that discolor and burn the skin. An initial yellowish-brown tanning turns to deep mahogany or purple-brown within a few hours of exposure. Blistering may follow in a day or two. Eye exposure may require medical treatment. A few species from the genera *Spirobolida*, *Spirostreptus*, and *Rhinocrichus* can squirt their secretions a distance of 80 cm or more.

5. Cimicidae (bed bugs).

There are over 90 species in the family Cimicidae. Most are associated with birds and/or bats and rarely bite humans. The common bed bug, *Cimex lectularius*, has been associated with humans for centuries and is cosmopolitan in distribution. The tropical bed bug, *Cimex hemipterus*, also feeds on humans and is similar in appearance to *C. lectularius*. It is common in tropical areas of Asia, Africa and Central America. Bed bug infestations are typical of unsanitary conditions, but they can still be found in developed countries. There is little evidence that bed bugs transmit any pathogens. Bites can be very irritating, prone to secondary infection after scratching, and may produce hard swellings or welts. Bed bugs feed at night while their hosts are sleeping but will feed during the day if conditions are favorable. During the day they hide in cracks and crevices, under mattresses, in mattress seams, spaces under baseboards, or loose wallpaper. Chronic exposure to bed bugs can result in insomnia, nervousness and fatigue. Some studies have found that a high percentage of asthmatic patients had positive skin reactions to *Cimex* antigen.

Five nymphal instars precede the adult stage. Each nymph must take a bloodmeal in order to molt. Adults live up to 1 year. Bed bugs take about 5 minutes to obtain a full bloodmeal. They can survive long periods of time without feeding, reappearing from their hiding places when hosts become available. Bed bugs possess scent glands and emit a characteristic odor that can easily be detected in heavily infested areas.

Blood spots on bedding or "bedclothes" and fecal deposits are other signs of infestation. Eggs and cast-off nymphal skins may be observed in cracks and crevices.

Infestations of bed bugs in human habitations are not uncommon in many areas of South Central Asia. Bed bugs can be introduced into barracks through infested baggage, bedding and belongings. They may pass from the clothing of one person to another on crowded public vehicles. In contingency situations, old dwellings should be surveyed for these and other pests before they are occupied. *Cimex lectularius* and *C. hemipterus* commonly feed on poultry in many parts of the region, so poultry houses should be avoided by military personnel.

6. Dipterans Causing Myiasis.

Myiasis refers to the condition of fly maggots infesting the organs and tissues of people or animals. Worldwide there are 3 major families of myiasis-producing flies: Oestridae, Calliphoridae and Sarcophagidae. The Oestridae contains about 150 species known as bot flies and warble flies. They are all obligate parasites, primarily on wild or domestic animals. Members of the genera *Cuterebra* and *Dermatobia* commonly infest humans in the Americas. The Calliphoridae, known as blow flies, are a large family composed of over 1,000 species. At least 80 species, mostly in the genera *Cochliomyia*, *Chrysomya*, *Calliphora* and *Lucilia*, have been recorded as causing cutaneous myiasis. Flies in the genus *Lucilia* are known as greenbottle flies due to their metallic or coppery green color. *Lucilia sericata* and *L. cuprina* are the most common species infesting wounds of humans. *Calliphora* flies are commonly called bluebottle flies because of their metallic-bluish or bluish-black color. The abdomen is usually more shiny than the thorax. The family Sarcophagidae, known as flesh flies, contains over 2,000 species, but its only important genera in terms of myiasis is *Wohlfahrtia* and *Sarcophaga*. The abdomen of flesh flies is often marked with squarish dark patches on a grey backgroundg, giving it a checkerboard appearance. Females are larviparous and deposit first-instar larvae instead of eggs. The larvae are deposited in batches of 40 to 60 on decaying carcasses, rotting food and animal or human feces.

Myiasis is also classified according to the type of host-parasite relationship, and specific cases of myiasis are clinically defined by the affected organ, e.g., cutaneous, enteric, rectal, aural, urogenital, ocular, etc. Myiasis can be accidental when fly larvae occasionally find their way into the human body. Accidental enteric myiasis occurs from ingesting fly eggs or young maggots on uncooked foods or previously cooked foods that have been subsequently infested. Other cases may occur from the use of contaminated catheters, douching syringes, or other invasive medical equipment in field hospitals. Accidental enteric myiasis is usually a benign event, but larvae may survive temporarily, causing stomach pains, nausea, or vomiting. Numerous fly species in the families Muscidae, Calliphoridae, and Sarcophagidae are involved in accidental enteric myiasis. A common example is the cheese skipper, *Piophila casei* (family Piophilidae), which infests cheese, dried meats and fish.

Facultative myiasis occurs when fly larvae infest living tissues opportunistically after feeding on decaying tissues in neglected wounds. Considerable pain and injury may be experienced as fly larvae invade healthy tissues. Facultative myiasis has been common in wounded soldiers throughout military history, and numerous species of Muscidae, Calliphoridae, and Sarcophagidae have been implicated. Species of these families are widespread throughout South Central Asia. Surgeons used maggots that feed only on necrotic tissue to clean septic battle wounds until about 1950. Maggot therapy has been used in recent years to treat chronically infected tissues, especially osteomyelitis.

Myiasis is obligate when fly larvae must develop in living tissues. This constitutes true parasitism and is essentially a zoonosis. Obligate myiasis is a serious pathology. In humans, obligate myiasis results primarily from fly species that normally parasitize domestic and wild animals. The sheep bot fly, *Oestrus ovis*, is found wherever sheep are raised. Larvae are obligate parasites in the nostrils and frontal sinuses of sheep, goats, camels and horses. Human ocular infestation by *O. ovis* is common in South Central Asia. Several cases occurred in US military personnel during the Persian Gulf War. Female flies are larviparous, depositing larvae while in flight directly into the human eye. Normally, infestations produce a painful but not serious form of conjunctivitis. However, larvae are capable of penetrating to the inner eye, causing serious complications.

The Old World screw-worm fly, *Chrysomya bezziana* (family, Calliphoridae), is a common myiasis-producing fly in South Central Asia. Adult *C. bezziana* only oviposit on live mammals, depositing 150 to 500 eggs at wound sites or in body orifices (ears, nose, mouth and urogenital openings). The larvae hatch in 18 to 24 hours, molt once after 12 to 18 hours, and a second time about 30 hours later. They feed for 3 to 4 days and drop to the ground to pupate. The pupal stage lasts 7 to 9 days in tropical conditions. *Chrysomya megacephala* is common in India and is often called the Oriental latrine fly because of its habit of breeding in feces as well as on carrion and other decomposing organic matter. It can occur in large numbers around latrines and become a nuisance in open-air meat and fish markets. The larvae can cause a secondary myiasis of wounds in man and animals.

Myiasis is rarely fatal, but troops living in the field during combat are at a high risk of infestation. Good sanitation can prevent most cases of accidental and facultative myiasis. To prevent flies from ovipositing on them, exposed foodstuffs should not be left unattended. Fruits and vegetables should be washed prior to consumption and examined for developing maggots. Extra care should be taken to keep wounds clean and dressed. Avoid sleeping in the nude, especially outdoors during daytime when adult flies are active and likely to oviposit in body orifices. At field facilities, proper waste disposal and fly control can reduce fly populations and the risk of infestation.

Several other species of flies commonly cause myiasis in cattle (e.g., *Hypoderma* spp.) And in horses and donkeys (e.g., *Gasterophilus* spp.), and their larvae sometimes infest humans. The larvae of most species of flies are extremely difficult to identify. Geographic location and type of myiasis are important clues to identity. It is particularly helpful to rear larval specimens so that the adult can be used for identification.

7. Hymenoptera (ants, bees and wasps).

Most wasps and some bees are solitary or subsocial insects that use their stings for subduing prey. These species are not usually involved in stinging incidents, and their venom generally causes only slight and temporary pain to humans. The social wasps, bees and ants use their sting primarily as a defensive weapon, and their venom causes intense pain in vertebrates.

The 3 families of Hymenoptera responsible for most stings in humans are the Vespidae (wasps, hornets, and yellow jackets), the Apidae (honey bees and bumble bees), and the Formicidae (ants). Wasps and ants can retract their stings after use and can sting repeatedly. The honey bee stinging apparatus has barbs that hold it so firmly that the bee's abdomen ruptures when it tries to pull the stinger out of the skin. The bee's poison gland, which is attached to the stinger, will continue injecting venom after separation. Scraping the skin after a bee sting is important to remove the stinger and attached venom sac. Honey bees and social wasps of the family Vespidae account for most stings requiring medical treatment in South Central Asia. Wild strains of honey bees may be more aggressive than domesticated populations maintained by bee keepers. *Apis cerana* is a common cavity-nesting honey bee that occurs in Asia, from Afghanistan to China and from Japan to southern Indonesia. The largest honey bees in the world, *Apis laboriosa* and *Apis dorsata*, are found in Central Asia. *Apis dorsata* occurs from Pakistan through the Indian Subcontinent and Sri Lanka to Indonesia and parts of the Philippines. The closely related, slightly larger *Apis laboriosa* is believed to be restricted to the Himalayas. The nests of both of these species are harvested by local people for honey and wax, and sometimes brood, which are eaten in Nepal and elsewhere. *Apis laboriosa* make its nests on the sides of cliffs, and *Apis dorsata* builds nests under tree limbs and cliffs. These bees will aggressively defend their nests and sometimes pursue attackers for over 100 meters. The world’s smallest honey bee, *Apis florae*, occurs from Oman, Iran and Pakistan through the Indian Subcontinent and Sri Lanka to Indonesia. *Apis florae* is most abundant in Southeast Asia, and it is not found north of the Himalayas.

Ants can bite, sting and squirt the contents of the poison gland through the tip of the abdomen as defensive secretions. The components of the venom are complex and vary with the species of ant. Formic acid is a common substance discharged as a defensive secretion. The Samsum ant, *Pachycondyla sennaarensis*, has been responsible for many hypersensitive reactions in Pakistan. Some protein-feeding ants such as the

Pharaoh ant, *Monomorium pharaonis*, have been incriminated as mechanical vectors of pathogens in hospitals.

Hymenoptera venoms have not been fully characterized but contain complex mixtures of allergenic proteins and peptides as well as vasoactive substances, such as histamine and norepinephrine. These are responsible for the pain at the sting site, irritation, redness of the skin, and allergic reactions in sensitized individuals. There is no allergic cross-reactivity between honey bee and vespid venoms, although cross-reactivity may exist to some extent between different vespid venoms. Therefore, a person sensitized to one vespid venom could have a serious reaction to the sting of another member of the vespid family.

Reactions to stings may be grouped into 2 categories, immediate (within 2 hours) or delayed (more than two hours). Immediate reactions are the most common and are subdivided into local, large local, or systemic allergic reactions. Local reactions are nonallergic responses characterized by erythema, swelling, and transient pain at the sting site that subsides in a few hours. Stings in the mouth or throat may require medical assistance. Multiple stings in a short period of time may cause systemic symptoms such as nausea, malaise and fever. It generally takes 500 or more honey bee stings to kill an adult by the toxic effects of the venom alone. The toxicity of African honey bee venom is roughly equivalent to the toxicity of the venom of domesticated honey bees. Large local reactions are characterized by painful swellings at least 5 cm in diameter and may involve an entire extremity. Systemic reactions vary from mild urticaria to more severe reactions, including vomiting, dizziness and wheezing. Severe allergic reactions are rare but can result in anaphylactic shock, difficulty in breathing, and death within 30 minutes. Emergency kits should be provided to patients who have experienced anaphylactic reactions to stings. Commercial kits are available that include antihistamine tablets and syringes preloaded with epinephrine. Sensitive individuals should also consider wearing a Medic-Alert tag to alert medical personnel of their allergy in case they lose consciousness. Venom immunotherapy for sensitive individuals will reduce but not eliminate the risk of anaphylactic reactions. The frequency of sting hypersensitivity is probably less than 1% of the population.

Delayed reactions to Hymenoptera envenomization are uncommon but usually present as a large local swelling or, rarely, as systemic syndromes. The cause of delayed reactions is unclear and may not always involve immunologic mechanisms.

Individuals can practice a number of precautions to avoid stinging insects. Avoid wearing brightly colored floral-pattern clothes. Do not go barefoot in fields where bees and wasps may be feeding at ground level. Avoid the use of scented sprays, perfumes, shampoos, suntan lotions, and soaps when working outdoors. Be cautious around rotting fruit, trash containers, and littered picnic grounds, since large numbers of yellow jackets often feed in these areas. Avoid drinking sodas or eating fruits and other sweets outdoors, since bees and yellow jackets are attracted to these items. Bees and wasps are most aggressive around their nests, which should not be disturbed.

8. Lepidoptera (urticating moths and caterpillars).

The caterpillars of certain moths possess urticating hairs that can cause dermatitis. The hairs are usually connected to glands that release poison when the hair tips break in human skin. The intensity of the irritation varies with the species of moth, sites and extent of exposure, and the sensitivity of the individual, but usually the symptoms are temporary. Hairs stimulate the release of histamine, and resultant skin rashes last about a week. The irritation is more severe when the hairs reach mucous membranes or the eye, where they can cause nodular conjunctivitis. Urticating hairs can also become attached to the cocoon when the larva pupates, and later to the adult moth. Hairs readily become airborne. If inhaled, detached caterpillar hairs can cause labored breathing; if ingested, they can cause mouth irritation. The hairs of some species retain their urticating properties long after being shed. Hairs and setae may drop into swimming pools and irritate swimmers.

Scratching and rubbing the affected parts of the body should be avoided to prevent venomous hairs from penetrating deeply into tissues. Running water should be used to wash the hairs out of the lesion. Light application of adhesive tape and stripping it away will remove many of the hairs or spines from the skin. Acute urticarial lesions usually respond to topical corticosteroid lotions and creams, which reduce the

inflammatory reaction. Oral histamines help relieve itching and burning sensations. Caterpillars of the families Bombycidae and Saturniidae have been implicated in cases of urticarial dermatitis in India and Sri Lanka.

9. Meloidae (blister beetles), Oedemeridae (false blister beetles) and Staphylinidae (rove beetles).

Blister beetles are moderate-sized (10 to 25 mm in length), soft-bodied insects that produce cantharidin in their body fluids. Cantharidin is a strong vesicant that readily penetrates the skin. Handling or crushing the beetles causes blistering within a few hours of skin contact. There is a large variation in individual susceptibility to blistering from cantharidin. Blisters are generally not serious and normally clear within 7 to 10 days without scarring. If blister beetles are ingested, cantharidin can cause nausea, diarrhea, vomiting, and abdominal cramps. Blisters that occur on the feet where they will be rubbed may need to be drained and treated with antiseptics. Cantharidin was once regarded as an aphrodisiac, and a European species of blister beetle was popularly known as Spanish-fly. Troops should be warned against using blister beetles for this purpose, since cantharidin is highly toxic when taken orally.

Approximately 1,500 species of Oedemeridae are found worldwide. They are slender, soft-bodied beetles, 5 to 20 mm in length. The adults of most species feed on pollen, so they are commonly found on flowers. These beetles also contain the vesicant cantharidin. Although there are few references in the medical literature, blister beetle dermatitis caused by oedemerids may be more common and widespread than currently recognized. During a training exercise on the North Island of New Zealand in 1987, 74 of 531 soldiers developed blistering after exposure to *Thelyphassa lineata*. Oedemerids are readily attracted to light.

The Staphylinidae, commonly called rove beetles, is another family that produces a strong vesicating substance that causes blistering. Rove beetles are active insects that run or fly rapidly. When running, they frequently raise the tip of the abdomen, much as scorpions do. They vary in size, but the largest are about 25 mm in length. Some of the larger rove beetles can inflict a painful bite when handled. Many species are small (<5 mm) and can get under clothing or in the eyes. Members of the genus *Paederus* are widespread throughout the world. They have a toxin, paederin, that can cause dermatitis, painful conjunctivitis and temporary blindness after eye contact. Normally, rove beetles must be crushed to release the vesicating agent. Like beetles in the family Meloidae, rove beetles are attracted to light and can be a hazard to soldiers at guard posts. Rove beetles often emerge in large numbers after rains and can cause outbreaks of dermatitis. A 1966 outbreak of blistering on Okinawa resulted in 2,000 people seeking medical treatment. During a 2-year period in the mid 1990s, 124 cases of vesicating dermatitis were reported among patients and staff of a modern hospital recently built in Sri Lanka. The staphylinid *Paederus fuscipes* was identified as the cause of the outbreak of dermatitis. Beetles were attracted by hospital lights at night and entered through open windows. *Paederus peregrinus* commonly causes dermatitis in eastern India.

10. Scorpionida (scorpions).

These arthropods have a stout cephalothorax, 4 pairs of legs, a pair of large anterior pedipalps with enlarged claws, and a tail tipped with a bulbous poison gland and stinger. Some species carry the tail above the dorsum of the thorax, while others drag it behind. Of over 1,500 described species worldwide, fewer than 25, all in the family Buthidae, possess a venom that is life threatening to humans. Scorpions inject the venom with a stinger on the tip of their abdomen, and some species can inflict a painful pinch with their pedipalps. They feed at night on insects, spiders and other arthropods. During the daytime, scorpions hide beneath stones, logs or bark, loose earth or among manmade objects. In dwellings, scorpions frequently rest in shoes or clothing.

Scorpions use their sting to capture prey, for defense against predators and during mating. The venom sacs are controlled voluntarily, so a scorpion can regulate how much venom is injected with each sting. Some scorpions may not inject any venom while stinging. Scorpion venom is a complex mixture of substances that may include several neurotoxins, histamine, seratonin, enzymes and other unidentified components. The venom of most species has never been analyzed. Some scorpion venoms are among the most toxic

substances known; fortunately, only a small amount is injected, probably less than 0.5 mg. There is evidence indicating that the toxicity of any species' venom is highly variable across its geographic range. Thus, a species that is dangerous in one area may not be hazardous in another.
There are numerous scorpions in South Central Asia. Afghanistan, India and Pakistan have many poisonous species, though fewer medically important scorpions occur in Nepal and Sri Lanka. Two medically important species, *Androctonus amoreuxi balcuchicus* and *Mesobuthus europus*, occur in Afghanistan and Pakistan. Also, *Mesobuthus tamulus tamulus*, the "Indian Red Scorpion," and *Ondotrobuthus ondonturus*, reported in India and Pakistan, are responsible for many scorpion stings. India has a number of medically important species that do not occur in the other South Central Asian countries, including the "Black or Burrowing Scorpion," *Heterometrus* (*Chersonesometrus*) *fulvipes*. Pakistan has both *M. europus* and *M. tamulus tamulus*, whose stings have been reported to cause death in small children. Some of the world's largest living scorpions (*Heterometrius* spp. and *Gigantometrus* spp.), which may reach lengths of 16 to 20 cm, are found in India and Sri Lanka. A list of scorpions reported from South Central Asia appears in Appendix A.5.

Most stings are to the lower extremities or the arms and hands. Among indigenous populations, stings are more often inflicted at night, while scorpions are actively hunting for prey. Scorpion stings can occur year-round in Pakistan, southern India, Bangladesh, the Maldives and Sri Lanka. However, in countries with colder climates (Afghanistan, Nepal and Bhutan), most stings are reported during the warmer months of March to October. In Pakistan, the sting of *Mesobuthus tamulus* or *M. europus* causes sharp localized pain, numbness in the affected limb, edema, occasional hospitalization, and even death in small children. No scorpions of significant medical importance have been reported from the Maldive islands.

Scorpions can sting multiple times, and when trapped, as with a person in a sleeping bag, will readily do so, as long as the victim is active. Common places where stings are encountered by military personnel include the boots and under or around piled clothing. Scorpion stings broadly affect nearly all body tissues, and they present a mixture of hemolytic, neurotoxic and cardiotoxic effects. All stings should be considered potentially dangerous. The severity of scorpion stings can be categorized as follows: 1) patients with initial sharp pain, numbness, and localized swelling dissipating in 1 to 3 hours with no systemic findings; 2) those who, in addition to pain, have 1 or 2 mild systemic manifestations, such as local muscle spasm, dry mouth, increased salivation, or runny nose; 3) those who have more severe systemic manifestations but no central nervous system manifestation or general paralysis; and 4) those who have severe systemic reactions, including central nervous system involvement, such as confusion, convulsions, and coma, with or without general paralysis. They may also develop uncoordinated eye movements, penile swelling, or cyanosis. The most severe manifestations occur in children, who are more susceptible to the effects of venom because of their small body mass. Those with type 1, 2, or 3 manifestations can be managed by applying ice to slow the spread of the venom, and supporting the patient with fluids and antihistamines. However, those with type 4 manifestations require intensive medical treatment, especially during the first 24 hours following the sting. Antivenin therapy is important for severe cases. For this treatment to be effective, the stinging scorpion must be captured so it can be properly identified.

To prevent scorpion stings, military personnel should be instructed to empty boots before attempting to put them on, carefully inspect clothing left on the ground before putting it on, and keep sleeping bags tightly rolled when not in use. Also, troops must be cautioned that scorpions can cause painful reactions requiring medical treatment and should never be kept or handled as pets.

11. Simuliidae (black flies, buffalo gnats, turkey gnats).

Black flies are small (3 to 5 mm), usually dark, stout-bodied, hump-backed flies with short wings. Despite their appearance, black flies are strong flyers that are capable of dispersing many kilometers from their breeding sites. Only females suck blood. They can emerge in large numbers and be serious pests of both livestock and humans. Black flies bite during the day and in the open. Some species have a bimodal pattern of activity, with peaks around 0900 hrs in the morning and 1700 hrs in the afternoon, but in shaded areas biting is more evenly distributed throughout the day. The arms, legs and face are common sites of attack, and a favorite site is the nape of the neck. Black fly bites may be itchy and slow to heal. Systemic reactions, characterized by wheezing, fever or widespread urticaria, are rare but require medical evaluation

and treatment. Numerous species of anthropophilic black flies are distributed throughout South Central Asia and have been a significant source of human discomfort.

12. Siphonaptera (fleas).

Fleabites can be an immense source of discomfort. The typical fleabite consists of a central spot surrounded by an erythematous ring. There is usually little swelling, but the center may be elevated into a papule. Papular urticaria is seen in persons with chronic exposure to fleabites. In sensitized individuals, a delayed papular reaction with intense itching may require medical treatment.

Fleas are extremely mobile, jumping as high as 30 cm. Biting often occurs around the ankles when troops walk through flea-infested habitat. Blousing trousers inside boots is essential to provide a barrier, since fleas will crawl under blousing garters. Fleas may be encountered in large numbers shortly after entering an abandoned dwelling, where flea pupae may remain in a quiescent state for long periods of time. The activity of anyone entering such premises will stimulate a mass emergence of hungry fleas. The most common pest fleas encountered in South Central Asia are the cosmopolitan cat and dog fleas, *Ctenocephalides felis* and *C. canis*, the Oriental rat flea, *Xenopsylla cheopis*, the related *X. astia*, and the human flea, *Pulex irritans*. A list of species reported from this region appears in Appendix A.4.

Tungan penetrans is also known as the chigoe, jigger, chigger, chique or sand flea. The chigoe is a tiny (about 1 mm long) that is native to South America. It was probably introduced into Africa in the seventeenth century and rapidly disseminated by expeditions throughout tropical Africa. Indian laborers returning to their homeland from Africa carried the parasite to Bombay, India and later to Karachi, Pakistan. The fertilized female cuts the host skin with her mouthparts and inserts her head and body until only the last 2 abdominal segments are exposed. The gravid female may become as large as a pea. Females feed and periodically deposit eggs. Most eggs fall to the ground and hatch, but a few may hatch within the nodular swelling of the host skin before falling to the ground. Larval development is completed within 2 weeks. *Tungan penetrans* usually attacks humans between the toes, under the toe nails and into the soles of the feet. Attached fleas cause extreme irritation, and the resulting nodules ulcerate and become infected. Ulcerations due to the presence of numerous chigoes may become confluent. Tetanus and gangrene may result from secondary infection, and autoamputation of toes has been recorded. *Tungan penetrans* will also other animals, especially swine. Where the chigoe occurs, walking in bare feet should be avoided. Attached fleas should be immediately removed with a sterile needle or fine-pointed tweezers.

13. Solpugida (sun spiders, wind scorpions).

These arthropods inhabit tropical and subtropical desert environments in Africa, Asia, Europe, and the Americas. One species, *Gylippus rickmersi*, has been reported from the Pamir plateau in Central Asia at an elevation of over 3,000 m. They usually avoid oases and other fertile places, seeming to prefer utterly neglected regions where the soil is broken and bare. Their hairy, spider-like appearance and ability to run rapidly across the ground account for their common names. Sun spiders range from 20 to 35 mm in body length and are usually pale colored. They have very large, powerful chelicerae, giving them a ferocious appearance. They can inflict a painful bite but do not have venom glands. Sun spiders are largely nocturnal, hiding during the day under objects or in burrows. They are aggressive and voracious predators on other arthropods. They easily kill scorpions and may even capture small lizards. At night they sometimes enter tents to catch flies or other insects.

14. Tabanidae (deer flies and horse flies).

Tabanids are large, stout-bodied flies with well-developed eyes that are often brilliantly colored. More than 4,000 species have been described worldwide. The larvae develop in moist or semiaquatic sites, such as the margins of ponds, salt marshes or damp earth. The immature stages are unknown for most species. Mature larvae migrate from their muddy habitats to drier areas of soil to pupate. Larval development is prolonged, and many species spend 1 to 2 years as larvae. In temperate regions the entire life cycle can take 2 years or more to complete. The larvae of horse flies are carnivorous and cannibalistic, whereas deer fly larvae feed on plant material. Consequently, deer fly populations can reach considerably higher

numbers in the same area. Carnivorous tabanid larvae occasionally bite humans, such as military personnel walking barefoot in rice fields or other areas containing such larvae. These bites can be quite painful.

Deer flies, about 8 to 15 mm long, are about half the size of horse flies, which range from 20 to 25 mm long. The most common tabanid genera containing man-biting species are *Chrysops* (deer flies), and *Tabanus* and *Haematopota* (horse flies). *Chrysops* and *Tabanus* have a worldwide distribution. *Haematopota* species, also known as clegs or stouts, are not found in South America or Australia, and only a few species occur in North America. However, they are common in Europe, Asia, Africa, India and the Far East.

Only female tabanids bite and take a bloodmeal, and nearly all species feed on mammals. Males feed on flower and vegetable juices. Tabanids are diurnal and are most active on warm, sunny days with low wind speeds, especially during the early morning and late afternoon. Adults are powerful flyers with a range of several kilometers. They are very persistent biters, and their painful bites are extremely annoying. They locate their hosts mainly by sight (color and movement), although olfactory stimuli like carbon dioxide and other host odors are involved.

Tabanids lacerate the skin with scissor-like mouthparts and ingest the blood that flows into the wound. Their mouthparts are large enough to penetrate many types of clothing. Some species can consume as much as 200 mg of blood. The puncture in the skin continues to ooze blood after the fly has fed. Tabanid bites often become secondarily infected, and systemic reactions may occur in hypersensitive individuals. The mouthparts and feeding behavior of tabanids are well suited to the mechanical transmission of blood-borne pathogens, and these flies have been incriminated in the transmission of tularemia. Because their bites are painful, tabanids are frequently disturbed while feeding and move readily from host to host. In South Central Asia, tabanids are not vectors of human disease but are serious pests of livestock and transmit several diseases of veterinary importance.

Tabanids are difficult to control. Larval control is impractical due to the difficulty in locating breeding places. Since larvae of most species live below the surface of the soil, insecticides would not penetrate the soil and vegetation and contact the immature stages. Similar problems are encountered in the control of ceratopogonid larvae. ULV aerosols are generally ineffective against adults. Localized control can be achieved around military encampments using a variety of simple traps. The skin repellent DEET is only moderately effective against these flies.

B. Venomous Snakes of South Central Asia

There are 47 species of venomous terrestrial snakes in South Central Asia, and 24 species of venomous sea snakes. Of these, however, only a small number are commonly encountered and likely to inflict a poisonous bite. For a complete list of venomous snakes and their distribution in South Central Asia, see Table 1. The families of venomous snakes present in the region are Crotalidae, Elapidae, Viperidae, Colubridae, and Hydrophiidae. Only the biology and distribution of the most important snakes from each family will be discussed .

1. Elapidae. The king cobra, *Ophiophagus hannah*, is the most feared of the cobras in the region. It normally achieves a length of 3.5 m to 4.3 m, and occasionally reaches 5.5 m. This olive-green snake, with a yellow throat, can raise its body one-third of its length off the ground. Unlike other cobras in the region, it does not have a hood that spreads. This snake hunts other snakes, primarily during the day. It inhabits jungle areas, mangrove swamps, and plains with heavy rainfall, and is most active during the monsoon season. The female lays 20 to 40 eggs in a nest of dead leaves and twigs and remains coiled around the nest for 60 to 90 days until the eggs hatch. The young are 0.5 m long and black when born. The king cobra's neurotoxic venom, while very potent, is not as poisonous as that of other venomous snakes. However, the large quantity of venom injected during the bite produces high mortality in people and large animals, including elephants. Despite its massive size and fierce reputation, the king cobra is not an aggressive snake. Despite its massive size and fierce reputation, the king cobra is not an aggressive snake. Much more deliberate in its actions than the smaller, excitable cobras (*Naja* spp.), it will quickly crawl away if given the opportunity. King cobras can be aggressive while defending a small territory around

their nest containing eggs.

Cobras with hoods include the diocellate cobra (*Naja n. naja*), the monocled cobra (*N. n. kaouthia*), and the aocellate cobra (*N. n. oxiana*). These smaller cobras are distinguished by the markings on their hoods. The monocled cobra has a single round spot, called the ocellus, on the hood. The diocellate cobra has two ocelli on its hood, and the aocellate cobra lacks an ocellus on its hood. None of these snakes are more than half the length of the king cobra but are likely to be more excitable. A cobra cannot strike upward, only downward and rather slowly compared to a viper. They usually lay their eggs in rat burrows, termite mounds, or other sheltered places. They feed at night on rats and mice.

Snake charming is a street entertainment in India, Bangladesh and Pakistan. Some snake charmers perform with fully functional snakes. Others protect themselves by removing the fangs, tying shut the venom ducts, or sewing the lips closed. Others keep their snakes intact but train them not to strike. The cobra follows the motion of the flute and are not charmed by the music.

The kraits are the next most prominent group of elapid snakes. They have a characteristic appearance of hexagonal scales and striking whitish or yellowish bands on the body. They are small snakes (1.0 to 1.7 m) and are usually mostly blue or black in color. The mouth and fangs are small. The shape of the tail ranges from pointed to blunt. Kraits are most active at night and hunt other snakes, rodents or lizards. These snakes are generally timid unless disturbed. Their venom is neurotoxic and sometimes fatal but, unlike cobras, there is no swelling or burning at the bite site. The most common snakes in this group are the common krait (*Bungarus caeruleus*), the banded krait (*B. fasciatus*), the many banded krait (*B. multicinctus*), and the Sri Lankan krait (*B. ceylonicus*). Of these kraits, the bite of *B. multicinctus* is most likely to be fatal.

Coral snakes are another group of elapid snakes that are closely related to kraits. They are small and often brightly colored. Their bodies are slender and often have a pinkish coral color on their bellies. The most prominent coral snakes are *Callophis melanurus* (slender coral snake), *C. nigrescens* (striped coral snake), and *C. beddomei* (Beddome's coral snake). Although their venom is very toxic, they have small mouths and short fangs, requiring them to chew the venom into their prey.

2. Viperidae. Snakes in the family Viperidae have fangs that are long and tuck into the roof of the mouth when not in use. There are 2 prominent venomous subfamilies in this region, the Viperinae, known as the true vipers, and the Crotalinae, the pit vipers. Unlike other snakes, the pit vipers have a heat-sensing pit located between the eye and the nostril. A third subfamily, Azemiopinae, is represented by just one species, *Azemiops feae*. The toxicity of this snake's venom to man is unknown. It is a relatively small snake, less than 1 m in length.

a. Subfamily Viperinae. Russell's viper (*Daboia russelli*) is the most well known in this group. This snake has a V-shaped marking on its head, pointing toward the front. The head is broad, flat and triangular. Body ranges up to 1.3 m in length and has 3 longitudinal rows of reddish-brown spots or rings, though only the dorsal row is complete. The body color varies among 3 different forms of this species, but the pattern of rings is consistent. Russell's viper inhabits plains and hills up to 1,000 m and prefers open plains and bushy or abandoned rocky areas, where it hunts lizards, toads, and small mammals at night. It is extremely aggressive during encounters with humans and frequently inflicts fatal wounds. The young are produced viviparously, 20 to 60 at a time. When threatened, this snake produces a strong hissing sound.

The saw-scaled viper (*Echis carinatus*) is also a prominent snake in this region. It is small, rarely exceeding 1 m in length. It occurs most often in dry and semi-arid areas and avoids forested sites. The overall color is greenish to sandy above and whitish on the undersides, with light or dark brown spots on its sides. The chain of spots is arranged in a zig-zag pattern. The head is oval and has a prominent trident marking. The scales on the side of the body are strongly keeled and produce a hissing sound as the snake moves across a surface. The tail is short and stubby. The female is viviparous, giving birth to 4 to 8 young at a time. This snake is less aggressive and less dangerous than Russell's viper; however, it often lies concealed in the sand with just its head noticeable and may strike prey or humans without warning.

The levantine viper (*Vipera lebetina*) is gray to pale brown above, with a whitish belly spotted brown. The head has a V-shaped mark similar to Russell's viper and a dark spot below the eye. The length varies from 1.3 - 1.7 m.

Eristocophis macmahonii is known from Afghanistan and Pakistan. This snake, which is less than 1 m in length, has a broad, flattened head, moderately stout body, and a short tail. Despite its small size, its bite has caused fatalities.

b. Subfamily Crotalinae. The pit vipers belong in the subfamily Crotalinae. *Hypnale hypnale* is a common viper, also called Merrem's hump-nosed viper, that is abundant in Sri Lanka. This snake is small and stout, ranging from 0.4 to 0.6 m in length. The head is flat and triangular, with a snout that ends in a hump. The tail is short and tapering. The dorsum is variable in color but most commonly is light brown, with yellow and a trace of red. Dark brown, oval spots punctuate either side of the spine along the body. These snakes are terrestrial, nocturnal, and aggressive when disturbed. They are often encountered in fruit, tea, or other agricultural plantations, usually remaining under shrubs or litter. Although their bites have never proved fatal, they can produce serious systemic effects that will make the victim ill for a prolonged period.

The green pit viper, *Trimeresurus trigonocephalus*, is a common snake in the region and is widely distributed. This species is generally green and ranges from 0.75 to 1.3 m in length. The head is broad and triangular, with a pit between the nostril and the eye. It prefers low trees and bushes but also inhabits outbuildings around human dwellings. Although it is a sluggish snake, the green pit viper can strike with alacrity, frequently vibrating its tail. The venom is rarely toxic enough to produce a systemic reaction.

3. Colubridae. The family Colubridae is very prominent in the region, most notably in Sri Lanka. These are rear-fanged snakes with grooves that help carry the venom. In most of these snakes, the venom is usually not fatal, although fatalities are known. Of the colubrids known from South Central Asia, only *Cerberus r. rhynchops*, the dog-faced water snake, produces a bite that requires treatment. This is a dull-colored snake with a pear-shaped head. It reaches about 0.6 to 1.2 m in length. This snake is an avid swimmer, preferring brackish water. It feeds on fish during the day and night. Favored habitats include mangrove swamps, lagoons, creeks, and estuaries. This species hisses and produces an offensive odor when provoked.

4. Hydrophiidae. Sea snakes are all venomous and possess fixed fangs like their close relatives the elapids. Sea snakes must come to the surface to obtain air. The nostrils are located dorsally on the snout and have valves that close to exclude water. Their bodies are laterally compressed, and their paddle-like tails make them excellent swimmers. Sea snakes can dive up to 160 m, although generally they inhabit much shallower water. Sea snakes feed on fish, crustaceans, or other marine life. Hydrophiids are so specialized for sea life that they are virtually helpless on land. They give birth to living young. Sea snakes are most numerous during the monsoon season in South Central Asia, when they may move into rivers and along the coast. They are commonly encountered by fishermen, who are frequently bitten as they handle fish in their nets.

The yellow-bellied sea snake, *Pelamis platurus*, is the most widely distributed of the sea snakes and frequently goes into freshwater rivers for extended periods, sometimes for months. It also is found at the surface of the open ocean as it drifts with currents. This snake requires tropical waters where the average monthly temperature is at least 20EC. Unlike most sea snakes, it frequently occurs in large numbers in water slicks along the sea surface where it preys upon fish. This species will frequently go into a feeding frenzy and bite anything in its surroundings. Skin shedding is frequent and may occur every 12 days. The venom of sea snakes is quite toxic, although people are rarely envenomated because close personal contact is necessary for these small-mouth snakes to inflict enough venom. Only about 3% of envenomated persons actually die. Death is caused by toxicity at the neuromuscular junction, resulting in respiratory failure.

The distribution of venomous terrestrial snakes in South Central Asia appears in Table 1. Sources of snake antivenoms are listed in Appendix D. For additional information on snakes and snakebite, contact DPMIAC. Also consult: Management of Snakebite in the Field, section IX A, by LTC Hamilton.

Table 1. Distribution of Venomous Snakes in South Central Asia (+ = Present; ? = Uncertain).

Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
COLUBRIDAE								
Balanophis ceylonicus								+
Cerberus r. rhynchops				**?**				+
R. subminiatus		+	+	+		+		
ELAPIDAE								
Bungarus bungaroides			+	+		+		+
B. ceylonensis								+
B. caeruleus sindanus		+		+		+	+	+
B. fasciatus				+			+	+
B. lividus				+			+	
B. niger				+				
B. walli				+				
Calliophis beddomei				+				
C. bibroni				+				
C. maclellandi				+		+		
C. melanurus				+				+
C. nigrescens				+				

Table 1. Distribution of Venomous Snakes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Disteira stokesi???				+				+
N. naja	+	+		+		+	+	+
Naja n. kaouthia		+	+	+		+		
N. n. oxiana	+			+			+	
Ophiophagus hannah		+		+			+	
VIPERIDAE								
Agkistrodon halys	+							
A. himalayanus				+		+	+	
A. hypnale				+				+
A. intermedius	+							
Azemiops feae				**?**				
Daboia russelii		+	+	+		+	+	+
Echis carinatus	+			+			+	+
E. multisquamatus	+						+	
E. c. sochureki	+	+		+			+	+
E. c. sinhaleyus								+
E. c. pyramidium	+			**?**				
Eristocophis macmahonii	+						+	

Table 1. Distribution of Venomous Snakes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Gloydius himalayanus	?	+	?	+		+	+	
Hypnale hypnale				+				+
H. nepa								+
Ovophis monticola			+	+		+		
Pseudocerastes persicus	+						+	
Trimeresurus albolabris			+	+		+		
T. erythrurus		+	+	+		+	+	
T. gramineus				+			+	
T. huttoni				+				
T. jerdonii				+			+	
T. macrolepis				+				
T. malabaricus				+				
T. mucrosquamatus		+		+				
T. popeorum				+				
T. purpureomaculatus				+			+	
T. stejnegeri			+	+		+		
T. strigatus				+				
T. trigonocephalus								+

Table 1. Distribution of Venomous Snakes in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
Vipera lebetina				+			+	
HYDROPHIIDAE								
Astrosia stokesii		+		+	+		+	+
Enhydrina schistosa		+		+	+		+	+
Hydrophis bituberculatus		+		+				+
H. caerulescens		+		+	+		+	+
H. cyanocinctus		**?**		+	+		+	+
H. fasciatus		+		+	**?**		**?**	+
H. lapemoides		**?**		+	**?**		+	**?**
H. mamillaris		+		+	+		+	+
H. melanosoma		**?**		**?**				**?**
H. nigrocinctus		+		+				+
H. obscurus				+				+
H. ornatus		+		+	+		+	+
H. spiralis		+		+	+		+	+
H. stricticolis				+				+
Kerilia jerdonii				+				+
Kolophis annandalei				**?**				

Table 1. Distribution of Venomous Snakes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Lapemis curtus				+	?		+	?
L. hardwickii				?				?
Laticauda colubrina		?		?				?
Microcephalophis cantoris		+		+	+		+	+
M. gracilis		+		+	+		+	+
Pelamis platurus		+		+	+		+	+
Praescutata viperina		+		+	+		+	+
Thalassophis viperinus							+	+

C. Medical Botany.

1. Plants that Cause Contact Dermatitis. Plant dermatitis is a problem of enormous magnitude. Categories of dermal injury caused by plants include mechanical injury, immediate or delayed contact sensitivity, contact urticaria, phototoxicity and photoallergy, primary chemical irritation, or some combination of these. Plants causing contact dermatitis in South Central Asia are listed in Table 2.

Parthenium hysterophorus, native to the Carribbean region, was accidentally introduced in 1956 to Maharashtra State on the west coast of India. The plant spread rapidly along canal banks, roads and railways and has become a major pest weed. Airborne contact dermatitis caused by *P. hysterophorus* has become a serious public health problem in India. Epidemics of allergic contact dermatitis involving several thousand cases have occurred, including some fatalities. Treatment is difficult, and even potent topical corticosteroids are relatively ineffective. Airborne plant hairs and pollen contain allergenic sesquiterpene lactones. During the dry season, mature plants die and crumble to a fine dust that is scattered by the wind.

Members of the *Rhus* group (poison ivy, oak, and sumac) are the most frequent causes of acute allergic contact dermatitis. About 70% of the US population is sensitive to urushiol in the sap of these plants. Any part of the skin surface of a sensitized individual may react upon contact with *Rhus* spp. Urushiol remains active for up to 1year and is easily transferred from an object to a person, so anything that touches poison ivy (clothing, tools, animal fur, sleeping bags) ban be contaminated with urushiol and cause poison ivy in a person who touches the object. Even smoke from burning plants can produce a severe allergic response. Barrier creams have been developed to prevent contact dermatitis in people sensitive to urushiol but are only partially effective. Allergy to poison ivy, oak and sumac may also mean a person is allergic to related plants, including cashews, pistachios, mangos and Chinese or Japanese lacquer trees.

The marking nut tree, *Semecarpus anacardium*, bears nuts containing a black juice that is used to mark laundry in India and Malaya. Indian laundry workers frequently develop a dermatitis from the oil of this nut. Dermatitis affected English officers and servicemen who wore marked garments while stationed in India during World War II. The marking nut liquid contains a pentadecylcatechol that is capable of cross-reacting with poison ivy urushiol.

Contact urticaria may result from immunological or nonimmunological host responses, although the latter is more common. Nettles, such as *Urtica* spp. and *Laportea* spp., are examples of plants that cause nonimmunological contact urticaria. These plants have hollow stinging hairs that inject a chemical after penetration of the skin. A burning sensation and pruritis occur almost immediately.

A number of cultivated plants of the carrot and rue families sensitize the skin to long-wave ultraviolet light (phytophotodermatitis). Within 6 to 24 hours of contact with the plant and exposure to sunlight or fluorescent light, the area of contact will selectively burn. In some cases, hyperpigmentation may persist for several months. In South Central Asia several *Heracleum* species contain phototoxic furcocoumarins.

Some plants contain primary chemical irritants that produce skin damage resembling that from contact with a corrosive acid. The reaction depends on the potency of the irritant. The most serious reactions involve the eye. *Daphne* spp. and *Euphorbia* spp. are examples of plants containing chemical irritants.

Mechanical injury by splinters, thorns, spines and sharp leaf edges can produce visual impairment or fungal and bacterial infections at the site of injury. Some dried seeds are hygroscopic and can cause severe discomfort due to swelling of the plant tissues when lodged in the auditory canal or other body cavity.

For additional information on plants causing dermatitis, contact DPMIAC.

Table 2. Plants that Cause Contact Dermatitis in South Central Asia.								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Abrus precatorius		+	+	+		+		+
Achillea spp.	+	+	+	+		+	+	
Aconitum spp.			+	+		+		
Actaea spp.			+	+		+		
Agave spp.				+				
Aleurites spp.		+		+				
Ammannia spp.		+		+				+
Anacardium occidentale		+	+	+		+		
Argermone spp.	+	+	+	+			+	+
Asclepias spp.		+	+	+			+	
Calophyllum inophyllum		+	+	+		+		
Calotropis spp.		+	+	+		+	+	+
Croton spp.	+	+	+	+		+	+	+
Daphne spp.		+	+	+		+		+
Datura spp.	+	+	+	+		+	+	+
Duranta erecta (*D. repens*)		+		+				
Euphorbia spp.	+	+	+	+		+	+	+
Excoecaria spp.		+		+				+

Table 2. Plants that Cause Contact Dermatitis in South Central Asia.								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Gnidia spp. (*Lasiosiphon* spp.)							+	+
Heracleum spp.			+	+		+		
Jatropha spp.	+	+	+	+			+	+
Laportea spp.		+		+				+
Mangifera spp.		+	+	+		+		+
Mucuna spp.		+		+				+
Parthenium hysterophorus				+				
Rhus spp. (*Toxicodendron* spp.)				+				
Ricinus communis	+	+	+	+		+	+	+
Sapium spp.		+		+				
Semecarpus spp.		+		+				+
Sterculia spp.		+		+				+
Tephrosia spp.		+		+			+	
Urtica spp.		+	+	+		+	+	

2. Systemic Toxicity from Ingestion of Plants. Most wild plants contain toxic components, and military personnel must be instructed not to consume local plants unless necessary for survival. Wild plants are difficult to identify, and poisonous plants can easily be mistaken for plants with parts safe to eat. Military personnel will be forced by necessity to consume wild plants during survival operations. To avoid accidental poisoning, they should be thoroughly trained to recognize common edible plants in the region. Local inhabitants may be knowledgeable about poisonous plants in the area.

The cashew nut, *Anacardium occidentale*, is extremely toxic if eaten uncooked, and the resin in the plant can cause severe dermatitis. The cashew nut shell, but not the kernel, contains a brown oily juice that is a contact allergen. Roasting the shell liberates irritating vapors. In India, where cashews are grown commercially, cashew nut dermatitis often affects thousands of workers.

Many plants have fruiting bodies or have attractive parts that appear edible. *Ricinus communis*, the castor oil plant, has highly ornamental, oval seeds. Castor oil is derived from the seeds and the plant may be grown commercially in some tropical areas of South Central Asia. All parts of the plant, especially the seeds, contain ricin, one of the world's most toxic substances. If the beans are swallowed whole, the hard coat prevents absorption and therefore inhibits poisoning, but 2 to 6 beans can be fatal to an adult if well chewed. One or 2 seeds can be fatal to a child.

Seeds of *Abrus precatorius* (known variously as rosary pea, precatory bean, prayer vine or crab's eye) possess one of the most powerful plant toxins known. One or two seeds, if thoroughly chewed, are capable of killing an adult human. The proteinaceous toxin, abrin, is similar in toxic effects to that of ricin. It is readily absorbed through the digestive tract and causes serious and often fatal clotting in the bloodstream. The attractive seeds are part scarlet-red and part shiny black. They may be used in making rosary necklaces or other costume jewelry in some countries, although this practice is illegal in the U.S.

Some military personnel may be tempted to consume plants because they are used locally for various purposes. Local lore may attribute medicinal qualities, psychotropic or aphrodisiac effects to native plants. Betel nut, *Areca catechu*, is chewed in many areas of South Central Asia. The plant contains an alkaloid, arecoline, that possesses numerous pharmacological properties. Significant illness can be associated with its use, including asthma exacerbation, cholinergic crisis, cardiac arrhythmias, acute psychosis, milk-alkali syndrome, and oropharyngeal tumors from long-term use. Sometimes a less moist form of betelquid lacking the betel leaf is mixed with tobacco; the mixture is called pan masala/gutkha. Medical personnel and combat commanders must be aware that some troops will be tempted to experiment with native plants. Military personnel should not chew on any part of an unfamiliar plant or use unfamiliar plants for fuel or cooking materials.

In most cases of poisoning, care is usually symptom driven. The age and medical condition of the patient influence toxic response and medical treatment. Special monitoring and specific drug therapy are indicated in some instances. Because life-threatening intoxications are rare, military medical personnel may have little experience in management of plant poisoning. It is inappropriate to assume that the toxicity exhibited by a single member of a genus will apply to all other species of that genus or that all toxic members of a genus will have similar effects. Most toxic plants, regardless of their ultimate effects, induce fluid loss through vomiting and diarrhea. This is important when military personnel are operating in hot, arid areas. Plant toxicity varies with the plant part, maturity, growing conditions, and genetic variation.

TG 196, Guide to Poisonous and Toxic Plants, provides information on toxic plants common in the US that also occur in other regions of the world. It includes a list of state and regional poison control centers. For additional information, contact DPMIAC.

IX. Selected References.

A. Military Publications*

1966. Poisonous snakes of the world, a manual for use by U.S. amphibious forces. NAVMED P-5099, BUMED, Department of the Navy, U.S. Gov. Print. Off., 212 pp.

1987. Technical Information Memorandum (TIM) 23. A concise guide for the detection, prevention and control of schistosomiasis in the uniformed services. AFPMB, 40 pp.

1991. Technical Guide (TG) 138. Guide to commensal rodent control. U.S. Army Environmental Hygiene Agency. 91 pp.

1991. Venomous snakes of the Middle East. AFMIC, Fort Detrick, MD. DST-1810S-469-91, 168 pp.

1992. TG 189. Procedures for the diagnostic dose resistance test kits for mosquitoes, body lice, and beetle pests of stored products. USAEHA, 39 pp.

1993. TIM 31. Contingency retrograde washdowns: cleaning and inspection procedures. AFPMB, 8 pp., Appendices A-H.

1994. TG 196. Guide to poisonous and toxic plants. USAEHA, 70 pp.

1995. TG 103. Prevention and control of plague. USACHPPM, 100 pp.

1995. TIM 40. Methods for trapping and sampling small mammals for virologic testing. AFPMB, 61 pp.

1995. Management of snakebite in the field. (unpublished document compiled by LTC Hamilton, filed as DPMIAC 162252).

1998. TIM 26. Tick-borne diseases: vector surveillance and control. AFPMB, 53 pp., Appendices A-J.

1998. Navy Medical Department Pocket Guide to Malaria Prevention and Control. 2nd ed. Technical Manual NEHC- TM6250.98-2.

1999. TIM 41. Protection from rodent-borne diseases. AFPMB, 59 pp., Appendices A-E.

1999. TIM 13. Ultra low volume dispersal of insecticides by ground equipment. AFPMB, 20 pp.

2000. TIM 24. Contingency pest management guide. 6th Edition, AFPMB, 122 pp.

2001. TIM 36. Personal protective techniques against insects and other arthropods of military significance. AFPMB, 43 pp., 4 Appendices, Glossary.

2001. TIM 6. Delousing procedures for the control of louse-borne disease during contingency operations. AFPMB, 31 pp.

* TIMs can be downloaded from the AFPMB website at: <www.AFPMB.org/pubs/tims/tims.htm>

B. Other Publications

Amerasinghe, F.P. 1995. Rice field breeding mosquitoes (Diptera: Culicidae) in a new irrigation project in Sri Lanka. Mosq.-borne Dis. Bull. 10: 1-7.

Amerasinghe, F.P. and N.B. Munasingha. 1988. A predevelopment mosquito survey in the Mahaweli Development Project area, Sri Lanka: immatures. J. Med. Entomol. 25: 286-294.

Amerasinghe, F.P. and N.B. Munasingha. 1988. A predevelopment mosquito survey in the Mahaweli Development Project area, Sri Lanka: adults. J. Med. Entomol. 25: 276-285.

Anuradha, S., N.P. Singh, S.N. Rizvi, S.K. Agarwal, R. Gur and M.D. Mathur. 1998. The 1996 outbreak of dengue hemorrhagic fever in Delhi, India. Southeast Asian J. Trop. Med. Public Health 29: 503-506.

Arsen'eva L.P. 1982. Natural focal zoonoses in Afghanistan. (Review of literature). Med. Parazitol. 51: 54 -59.

Ayaz, M., A. Bari and A. Humayun. 1993. Coxiellosis in man and animals in northern parts of Pakistan. Proc. Pakistan Congr. Zool. 13: 425-431.

Beaty, B.J. and W.C. Marquardt [eds.]. 1996. The biology of disease vectors. University of Colorado Press. Niwot, CO.

Bhat, H. 1985. Observations on the biology of *Haemaphysalis spingera* Neumann (Acarina: Ixodidae) under natural conditions in KFD area. J. Bombay Natural History Soc. 82: 548-562.

Bishnupada, R. and V. Tandon. 1992. Louse infestations in human populations in Shillong, India. Health & Hygiene 13: 15-20.

Biswas, D., S. Dey, R.N. Dutta and A.K. Hati. 1993. Observations on the breeding habitats of *Aedes aegypti* in Calcutta following an episode of dengue hemorrhagic fever. Indian J. Med. Res. 97: 44-46.

Brogdon W.G. and J.C. McAllister. 1998. Insecticide resistance and vector control. Emerg. Infect. Dis. 4: 605-613.

Bruce-Chwatt, L.J. 1985. Essential malariology, 2nd ed., John Wiley and Sons, New York.

Bryan, J.P., M. Iqbal, T.G. Ksiazek, A. Ahmed, J.F. Duncan, B. Awan, R.E. Krieg, M. Riaz, J.W. Leduc, S. Nabi, M.S. Qureshi, I.A. Malik, and L.J. Legters. 1996. Prevalence of sand fly fever, West Nile, Crimean-Congo hemorrhagic fever, and leptospirosis antibodies in Pakistani military personnel. Military Med. 161: 149-153.

Chan, Y.C., N.I. Salahuddin, J. Khan, H.C. Tan, C.L.K. Seah, J. Li and V.T.K. Chow. 1995. Dengue hemorrhagic fever outbreak in Karachi, Pakistan. Trans. R. Soc. Trop. Med. Hyg. 89: 619-620.

Chand, S.K., R.S. Yadav and V.P. Sharma. 1993. Seasonality of indoor resting mosquitoes in a broken-forest ecosystem of north-western Orissa. Indian J. Malariol. 30: 145-154.

Chin, J. 2000. Control of communicable diseases manual. 17th ed., American Public Health Association, Washington, DC. (US Army FM 8-33, US Navy P-5038).

Crump, A. 1989. The perils of water-resource development: Japanese encephalitis. Parasitology Today 5: 343-344.

Dar, L., S. Sengupta, I. Xess and P. Seth. 1999. The first major outbreak of dengue hemorrhagic fever in Delhi, India. Emerg. Infect. Dis. 5: 589-590.

Darwish, M.A., H. Hoogstraal, T.J. Roberts, I.P. Ahmed and F. Omar. 1983. A sero-epidemiological survey for certain arboviruses (Togoviridae) in Pakistan. Trans. R. Soc. Trop. Med. Hyg. 77: 442-445.

Defraites, R.F., J.M. Gambel, C.H. Hoke Jr, J.L. Sanchez, B.G. Withers, N. Karabatsos, R.E. Shope, S. Tirrell, I. Yoshida, M. Takagi, C.K. Meschievitz and T.F Tsai. 1999. Japanese encephalitis vaccine (inactivated, BIKEN) in U.S. soldiers: immunogenicity and safety of vaccine in two dosing regimens. Am. J. Trop. Med. Hyg. 61: 288-293.

Dev, V. 1996. *Anopheles minimus*: its bionomics and role in the transmission of malaria in Assam, India. Bull. W.H.O. 74: 125-153.

Dhanda, V., P.K. Das, R. Lal, R. Srinivasan and K.D. Ramaiah. 1996. Spread of lymphatic filariasis, re-emergence of leishmaniasis and the threat of babesiosis in India. Indian J. Med. Res. 104: 46-54.

Dileep, V. and M.D. Mavalankar. 1995. Indian plague epidemic: unanswered questions and key lessons. J. Roy. Soc. Med. 88: 547-551.

Dutta, P., S.A. Khan, C.K. Sharma, P. Doloi and J. Mahanta. 1999. Medically important mosquitoes of the world's largest river island, Majuli, Assam. Entomon 24: 33-39.

Dutta, P., S.A. Khan, C.K. Sharma, P. Doloi, N.C. Hazarika and J. Mahanta. 1998. Distribution of potential dengue vectors in major townships along the national highways and trunk roads of northeast India. Southeast Asian J. Trop. Med. Public Health. 29: 173-176.

Elias, M. 1996. Larval habitat of *Anopheles philippinensis*: a vector of malaria in Bangladesh. Bull. W.H.O. 74: 447-450.

Elias, M., A.J.M.M. Rahman and N. I. Khan. 1989. Visceral leishmaniasis and its control in Bangladesh. Bull. W.H.O. 67: 43-49.

Gaidamovich, S.Y., N.V. Khutoretskaya, Y.V. Asyamov, V.I. Tsyupa and E.E. Melnikova. 1990. Sandfly fever in Central Asia and Afghanistan. Arch. Virol. 34: 287-293.

Gangadhar, N.L. and M. Rajasekhar. 1998. Record of rodent as natural reservoir host of *Leptospira javanica* in Karnataka. Indian Vet. J. 75: 563-564.

Gajanana, A., R. Rejendran, P.P. Samuel, V. Thenmozhi, T.F. Tsai, J. Kimura-Kuroda and R. Reuben. 1997. Japanese encephalitis in South Arcot District, Tamil Nadu, India: a three-year longitudinal study of vector abundance and infection frequency. J. Med. Entomol. 34: 65-659.

Geevarghese, G., A.C. Mishra, P.G. Jacob and H.R. Bhat. 1994. Studies on the mosquito vectors of Japanese encephalitis virus in Mandya District, Karnataka, India. Southeast Asian J. Trop. Med. Public Health. 25: 378-382.

Geevarghese, G., B.H. Shaikh, P.G. Jacob, H.R. Bhat and K.M. Pavri. 1987. Domestic pigs as sentinels to monitor the activity of Japanese encephalitis and West Nile fever viruses in Kolar District, Karnataka. Indian J. Med. Res. 86: 413-418.

Goddard, J. 1996. Physicians guide to arthropods of medical importance. 2nd ed., CRC Press, Inc., Boca Raton, FL.

Goverdhan, M.K., A.B. Kulkarni, A.K. Gupa, C.D. Tupe and J.J. Rodrigues. 1992. Two-way cross-protection between West Nile and Japanese encephalitis viruses in bonnet macaques. Acta Virologica 36: 277-283.

Grogl, M., J.L. Daugirda, D.L. Hoover, A.J. Magil and J.D. Berman. 1993. Survivability and infectivity of viscerotropic *Leishmania tropica* from Operation Desert Storm participants in human blood products maintained under blood bank conditions. Am. J. Trop. Med. Hyg. 49: 308-315.

Harwood, R.F. and M.T. James. 1979. Entomology in human and animal health. 7th ed., MacMillan Publishing Company, Inc., New York.

Hassan, K., N. Ikram, K.P. Bukhari, S.H. Shah and M. Hassan. 1995. Visceral leishmaniasis - a study of 38 cases on the basis of geographical distribution. J. Pak. Med. Assoc. 45: 125-127.

Hawley, W.A. 1988. The biology of *Aedes albopictus.* J. Amer. Mosq. Control Assoc.. Suppl. No. 1. pp 1-40.

Hoke, C. and J. Gingrich. 1994. Japanese encephalitis, pp 59-69. *In*: G. Bergan (ed.) Handbook of Zoonoses, Section B: Viral. CRC Press, Boca Raton.

Hoogstraal, H. 1979. The epidemiology of Crimean-Congo hemorrhagic fever in Asia, Europe, and Africa. J. Med. Entomol. 15: 307-417.

Hoogstraal, H. 1981. Changing patterns of tickborne diseases in modern society. pp. 75-100 *In*: Annual Review of Entomology, Vol. 26, Annual Reviews, Inc., Palo Alto, CA.

Hubalek, Z. 1987. Geographical distribution of Bhanja virus. Folia Parasitol. 34: 77-86.

Jamaluddain, M. and V.K. Saxena. 1997. First outbreak of dengue hemorrhagic fever in a typical rural area of Haryana State in northern India. J. Commun. Dis. 29: 169-170.

John, T.J. 1996. Emerging and re-emerging bacterial pathogens in India. Indian J. Med. Res. 103: 4-18.

Joshi, V., M. Singhi and R.C. Chaudhary. 1996. Transovarial transmission of dengue 3 virus by *Aedes aegypti*. Trans. R. Soc. Trop, Med. Hyg. 90: 643-644.

Joshi, V., M.L. Mathur, A.K. Dixit and M. Singhi. 1996. Entomological studies in a dengue area, Jalore, Rajasthan. Indian J. Med. Res. 104: 161-165.

Joshi, V., M. Singhi and R.C. Chaudhary. 1996. Transovarial transmission of dengue 3 by *Aedes aegypti*. Trans. R. Soc. Trop. Med. Hyg. 90: 643-644.

Kader, M.S.A., P. Kandaswamy, N.C. Appavoo and A. Anuradha. 1997. Outbreak and control of dengue in a village in Dharmapuri, Tamil Nadu. J. Commun. Dis. 29: 69-71.

Kahl, O., C. Janetzki-Mittmann, J.S. Gray, R. Jonas, J. Stein and R. de Boer. 1998. Risk of infection with *Borrelia burgdorferi* sensu lato for a host in relation to the duration of nymphal *Ixodes ricinus* feeding and the method of tick removal. Zentrabl. Bakteriol. 287: 41-52.

Kaliwal, M.B. and S.B. Nadkarni. 1992. Mosquito fauna in the irrigated areas of North Goa District, Goa. Indian J. Parasitol. 16: 35-38.

Kalyan, B. and K. Banerjee. 1996. Emerging viral infections with special reference to India. Indian J. Med. Res. 103: 177-200.

Kamimura, K., T. Takasu, Altaf-Ahmed and Aktar-Ahmed. 1986. A survey of mosquitoes in Karachi area, Pakistan. J. Pakistan Med. Assoc. 36: 182-188.

Karunaratne, S.H.P.P. and F.P. Amerasinghe. 1994. Ecology of four potential *Culex* vectors (Diptera: Culicidae) of Japanese encephalitis in Kandy, Sri Lanka. Ceylon J. Sci. 23: 38-46.

Kettle, D.S. [ed.]. 1995. Medical and veterinary entomology. 2nd ed., CAB International, University Press, Cambridge, UK.

Khan, A.M., A.Q. Khan, L. Dobrzynski, G.P. Joshi and A. Myat. 1981. A Japanese encephalitis focus in Bangladesh. J. Trop. Med. Hyg. 84: 41-44.

Khan, S.A., R. Handique, S.C. Tewari, P. Dutta, K. Narain and J. Mahanta. 1998. Larval ecology and mosquito fauna of Upper Brahmaputra Valley, northeast India. Indian J. Med. Res. 25: 131-145.

Khan, S.A., K. Narain, P. Dutta, R. Handique, V.K. Srivastava and J. Mahanta. 1997. Biting behavior and biting rhythm of potential Japanese encephalitis vectors in Assam. J. Commun. Dis. 29: 109-120.

Khan, S.A., K. Narain, R. Handique, P.Dutta, J. Mahanta, K. Satyanarayana and V.K. Srivastava. 1996. Role of some environmental factors in modulating seasonal abundance of potential Japanese encephalitis vectors in Assam, India. Southeast Asian J. Trop. Med. Public. Health. 27: 382-391.

Krishnamoorthy, K., G. Rajendran and K.N. Panicker. 1994. Aquatic vegetation and their natural hospitability to the immatures of *Mansonia* mosquitoes, the vectors of *Brugia malayi* in Shertallai, Kerala, India. Southeast Asian J. Trop. Med. Public Health 25; 760-765.

Kubo, T., S.K. Rai, S. Rawal and T. Yamano. Serological study of Japanese encephalitis in Nepal. Southeast Asian J. Trop. Med. Public Health 24: 756-761.

Kulkarni, S.M. 1987. Feeding behavior of anopheline mosquitoes in an area endemic for malaria in Bastar District, Madhya Pradesh. Indian J. Malariol. 24: 163-171.

Kulkarni, S.M., K. Banerjee, P.K. Deshmukh, M.A. Ilkal and N.C. Venkateshan. 1977. Dengue epidemic in Amalner Town, Jalgaon District, Maharashtra, India - entomological investigations. J. Commun. Dis. 9: 124-127.

Kumar, N., S. Sabesan and K. Panicker. 1989. Biting rhythm of the vectors of Malayan filariasis, *Mansonia annulifera*, *Ma. uniformis,* and *Ma. indiana*, the vectors of filariasis in Shertallai (Kerala State). Indian J. Med. Res. 89: 52-55.

Kumar, N., S. Sabesan and K. Panicker. 1992. The resting and house-frequenting behavior of *Mansonia annulifera*, *Ma. uniformis,* and *Ma. indiana*, the vectors of filariasis in Kerala State, India. Southeast Asian J. Trop. Med. Public Health 23: 325-327.

Kuriakose, M., C.K. Eapen and R. Paul. 1997. Leptospirosis in Kolenchery, Kerala, India: epidemiology, prevalent local serogroups and serovars and a new serovar. Eur. J. Epidemiol. 13: 691-697.

Laird, M. 1988. The natural history of larval mosquito habitats. Academic Press, New York.

Lacey, L.A. and C.M. Lacey. 1990. The medical importance of riceland mosquitoes and their control using alternatives to chemical insecticides. J. Amer. Mosq. Control Assoc., Suppl. No. 2, pp 1-93.

Lane, R.P. and R.W. Crosskey (eds.). 1993. Medical insects and arachnids. Chapman and Hall, London, UK.

Lane, R.P., M.M. Pile and F.P. Amerasinghe. 1990. Anthrophagy and aggregation behavior of the sandfly *Phlebotomus argentipes* in Sri Lanka. Med. Vet. Entomol. 4: 79-88.

Lawyer, P.G. and P.V. Perkins. 2000. Leishmaniasis and trypanosomiasis. Chapter 8 *In*: Medical Entomology. B.F. Eldridge and J.D. Edman (eds.).

Linthicum, K.J. and C.L. Bailey. 1994. Ecology of Crimean-Congo hemorrhagic fever, pp 392-437. *In*: Ecological Dynamics of Tick-borne Zoonoses, D. E. Sonenshine and T.N. Mather (eds). Oxford University Press.

Lovell, C. R. (ed.). 1993. Plants and the skin. Blackwell Scientific Publications, Oxford.

Mahadev, P.V.M. and M.D. Gokhale. Water storage and *Aedes aegypti* breeding in multistoreyed flats: a case study in Pune, Maharashtra State, India. Entomon 23: 173-183.

Mahadev, P.V.M., S.R. Prasad, M.A. Ilkal, M.S. Mavale, S.S. Bedekar and K. Banerjee. 1997. Activity of dengue-2 virus and prevalence of *Aedes aegypti* in the Chirimiri colliery area, Madhya Pradesh, India. Southeast Asian J. Trop. Med. Public Health 28: 126-137.

Mills, J.N., J.E. Childs, T.G. Ksiazek, C.J. Peters and W.M. Velleca. 1995. Methods for trapping and sampling small mammals for virological testing. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Atlanta, GA.

Minter, D.M. 1989. The leishmaniases. WHO/VBC/89.967 pp. 93-106.

Monath, T.P. (ed.). 1988/89. The arboviruses: epidemiology and ecology. Volumes I-V, CRC Press, Boca Raton, FL.

Morshed, M.G., H. Konishi, Y. Terada, Y. Arimitsu and T. Nakazawa. 1994. Seroprevalence of leptospirosis in a rural flood prone district of Bangladesh. Epidemiol. Infect. 112: 527-531.

Murhekar, M.V., A.P. Sugunan, P. Vijayachari, S. Sharma and S.C. Sehgal. 1998. Risk factors in the transmission of leptospiral infection. Indian J. Med. Res. 197: 218-223.

Narain, K., J. Mahanta, R. Dutta and P. Dutta. 1994. Paddy field dermatitis in Assam: a cercarial dermatitis. J. Commun. Dis. 26: 26-30.

Natarajaseenivasan, K. and S. Ratnam. 1997. Seroprevalence of leptospiral infection in an agricultural based village in Tamil Nadu. Cheiron 26: 80-83.

Neogi, D.K., N. Bhattacharya, K.K. Mukherjee, M.S. Chakraborty, M.K. Banerjee, K. Mitra, M. Lahiri and S.K. Chakravarti. 1995. Serosurvey of chikungunya antibody in Calcutta metropolis. J. Commun. Dis. 27: 19-22.

Nelson, B.S. and B. Heischober. 1999. Betel nut: a common drug used by naturalized citizens from India, Far East Asia, and the South Pacific Islands. Ann. Emerg. Med. 34: 238-243.

Nicholls, D.S.H., T.I. Christmas and D.E. Greig. 1990. Oedemerid blister beetle dermatosis: a review. J. Amer. Acad. Dermatol. 22: 815-819.

Nuttall, P.A. and M. Labuda. 1994. Tick-borne Encephalitis Subgroup. pp. 356-391. *In*: Ecological Dynamics of Tick-Borne Zoonoses, D.A. Sonenshine and T. N. Mather (eds). Oxford University Press.

Padbidri, V.S., J.J. Rodrigues, P.S. Shetty, M.V. Joshi, B.L. Rao and R.N. Shukla. 1984. Tick-borne rickettsioses in Pune District, Maharashtra, India. Int. J. Zoonoses 11: 45-52.

Panicker, K.N., M.G. Bai and M. Kalyanasundaram. 1982. Well breeding behavior of *Aedes aegypti*. Indian J. Med. Res. 76: 689-691.

Peiris, J.S.M., F.P. Amerasinghe, P.H. Amerasinghe, C.B. Ratnayake and S.H.P.P. Karunaratne. 1992. Japanese encephalitis in Sri Lanka - the study of an epidemic: vector incrimination, porcine infection and human diseases. Trans. R. Soc Trop. Med. Hyg. 86: 307-313.

Peiris, J.S.M., P.H. Amerasinghe, F.P. Amerasinghe, C.H. Calisher, L.P. Perera, C.K. Arunagiri, N.B. Munasingha and S.H. Karunaratne. 1994. Viruses isolated from mosquitoes collected in Sri Lanka. Am. J. Trop. Med. Hyg. 51: 154-161.

Peiris, J.S.M., F.P. Amerasinghe, C.K. Arunagiri, L.P. Perera, S.H.P.P. Karunaratne, C.B. Ratnayake, T.A. Kulatilaka and M.R.N. Abeysinghe. 1993. Japanese encephalitis in Sri Lanka: a comparison of vector and virus ecology in different agro-climatic areas. Trans. R. Soc. Trop. Med. Hyg. 87: 541-548.

Rajagopal, R. 1985. Studies on malaria in Bhutan. J. Commun. Dis. 17: 278-286.

Rajendran, G., K.N. Panicker, K. Krishnamoorthy, S. Sabesan, M. Snehlatha and R. Radhakrishnan. 1997. Current status of filariasis in Chavakad taluk, Trichur District Kerala. J. Commun. Dis. 29: 333-343.

Ram, S., S. Khurana, V. Kaushal, R. Gupta and S.B. Khurana. 1998. Incidence of dengue in relation to climatic factors in Ludhiana, Punjab. Indian J. Med. Res. 108: 128-133.

Ramaiah, K.D. and P.K. Das. 1992. Seasonality of adult *Culex quinquefasciatus* and transmission of bancroftian filariasis in Pondicherry, south India. Acta Trop. 50: 275-283

Ramakrishman, P. and V.N.A. Rao. 1987. A survey on Q fever in livestock. Cheiron 16: 109-111.

Ramasamy, M.S., R. Kulasekera, K.A. Srikrishnaraj and R. Ramasamy. 1994. Population dynamics of anthropophilic mosquitoes during the northeast monsoon season in the malaria epidemic zone of Sri Lanka. Med. Vet. Entomol. 8: 265-274.

Rana, U.V., S. Sehgal, R. Bhatia and M. Bhardwaj. 1987. Antibody against *Coxiella burnetii* in animals and humans in Delhi. J. Commun. Dis. 19: 152-155.

Raoult, D. and V. Roux. 1999. The body louse as a vector of reemerging human diseases. Clinical Infectious Diseases. 29: 888-911.

Ratnam, S., C.O. Everad, J.C. Alex, B. Suresh and P. Thangaraju. 1993. Prevalence of leptospiral agglutinins among conservancy workers in Madras City, India. J. Trop. Med. Hyg. 96: 41-45.

Ratnam, S., C.O. Everad and J.C. Alex. 1995. A pilot study on the prevalance of leptospirosis in Tamilnadu State. Indian Vet. J. 71: 1059-1963.

Reisen, W.K., F. Mahmood and T. Parveen. 1982. Seasonal trends in population size and survivorship of *Anopheles culicifacies*, *An. stephensi*, and *An. subpictus* in rural Punjab Province, Pakistan. J. Med. Entomol. 19: 86-97.

Reisen, W.K., S.P. Pradhan, J.P. Shrestha, S.L. Shrestha, R.G. Vaidya and J.D. Shrestha. 1993. Anopheline mosquito ecology in relation to malaria transmission in the inner and outer Terai of Nepal, 1987-1989. J. Med. Entomol. 30: 664-682.

Renapurkar, D.M. 1988. Observations on commensal rats and their status to plague in Bombay. J. Hyg. Epidemiol. Microbiol. Immunol. 32: 407-413.

Reuben, R., H. Kaul and R. Soman. 1988. Mosquitoes of arboviral importance in India. Mosq.-borne Dis. Bull. 5: 48-54.

Reuben, R., V. Thenmozhi, P.P. Samuel, A. Gajanana and T.R. Mani. 1992. Mosquito blood feeding patterns as a factor in the epidemiology of Japanese encephalitis in southern India. Amer. J. Trop. Med. Hyg. 46: 654-663.

Roberts, D.R. and R.G. Andre. 1994. Insecticide resistance issues in vector-borne disease control. Am. J. Trop. Med. Hyg. Suppl. 50: 21-34.

Rose, S.T. 1998. International travel health guide, 9th ed. Travel Medicine, Inc., Northampton, MA.

Sabesan, S., N.P. Kumar, K. Krishnamoorthy and K.N. Panicker. 1991. Seasonal abundance and biting behaviour of *Mansonia annulifera*, *M. uniformis* and *M. indiana* and their relative role in the transmission of Malayan filariasis in Shertallai (Kerala State). Indian J. Med. Res. 93: 253-258.

Salafsky, B., K. Ramaswamy, Y.X. He, J. Li and T. Shibuya. 1999. Development and evaluation of LIPODEET, a new long-acting formulation of N, N diethyl-m-toluamide (DEET) for the prevention of schistosomiasis. Am. J. Trop. Med. Hyg. 61: 743-750.

Samarawickrema, W.A., N. Jayasekera, C.G. Jansen, R.V. Chelliah, F.R. Karandawala and S. Pathmanathan. 1982. Significance of coconut husk pits as larval habitats of *Culex quinquefasciatus* (Say) in the filariasis endemic coastal belt of Sri Lanka. Southeast Asian J. Trop. Med. Public Health 13: 590-595.

Schmaljohn, C. and B. Hjelle. 1997. Hantaviruses: a global disease problem. Emerg. Infect. Dis. 3: 95-104.

Seal, S.C. 1988. History of researches on plague leading to its eradication in India. Proc. Indian Natl. Sci. Acad. 54: 215-238.

Sehgal, S.C., P. Vijayachari, M.V. Murhekar, A.P. Sugunan, S. Sharma and S.S. Singh. 1999. Leptospiral infection among primitive tribes of Andaman and Nicobar Islands. Epidemiol. Infect. 122: 423-428.

Seneviratne, J.K.K., S. Siriwardena, N. Ratnatunga and M. de S. Wijesundera. 1995. Locally acquired cutaneous leishmaniasis from central Sri Lanka: a case report. Kandy Med. J. 4: 54-56.

Service, M.W. 1995. Mosquito Ecology: Field Sampling Methods. 2nd Edition. Chapman and Hall, London.

Sexton, D.P. and H.P. Willett. 1992. Rickettsiae. pp. 700-718. *In*: Zinsser Microbiology, W.K. Joklik, H.P. Willett, D.B. Amos and C.M. Wilfert (eds.). 20th ed., Appleton and Lange, Norwalk, CT.

Shafi, M., R. Ali, R. Ghazi and N. Un-Nisa. 1988. Flea index studies of synanthropic rats in Karachi, Pakistan. Acta Parasitol. Polonica 33: 185-194.

Shah, I., M. Rowland, P. Mehmood, C. Mujahid, F. Razique, S. Hewitt and N. Durrant. 1997. Chlorquine resistance in Pakistan and the upsurge of falciparum malaria in Pakistani and Afghan refugee populations. Ann. Trop. Med. Parasitol. 91: 591-602.

Sharma, V.P. 1996. Ecological changes and vector-borne diseases. Trop. Ecol. 37: 57-65.

Sharma, S., M. Sharma and S. Rathaur. 1999. Bancroftian filariasis in the Varanasi region of north India: an epidemiological study. Ann. Trop. Med. Parasitol. 93: 379-387.

Sharma, R.S., R.S. Mishra, D. Pal, J.P. Gupta, M. Datta and K.K. Datta. 1984. An epidemiological study of scabies in a rural community in India. Ann. Trop. Med. Parasitol. 78: 157-164.

Sharma, S.K., N. Nanda, V.K. Dua, H. Joshi, S.K. Subbarao and V.P. Sharma. 1995. Studies on the bionomics of *Anopheles fluviatilis* sensu lato and the sibling species composition in the foothills of Shiwalik Range (Uttar Pradesh) India. Southeast Asian J. Trop. Med. Public Health 26: 566-572.

Sherchand, J.B., H. Ohara, S. Sherchand and H. Matsuda. 1999. The suspected existence of *Schistosoma mansoni* in Dhanusha District, southern Nepal. Ann. Trop. Med. Parasitol. 93: 273-278.

Shrestha, S.I. and S.K. Paul. 1994. Seasonal distribution of phlebotomine sandfly vectors of visceral leishmaniasis. J. Nepal Med. Assoc. 32: 237-246.

Silva, A.M. de, W.P. Dittus, P.H. Amerasinghe and F.P. Amerasinghe. 1999. Serological evidence for an epizootic dengue virus infecting toque macaques (*Macaca sinica*) at Polonnaruwa, Sri Lanka. Am. J. Trop. Med. Hyg. 60: 300-306.

Soman, R.S., D.T. Mourya and A.C. Mishra. 1986. Transovarial transmission of Japanese encephalitis virus in *Culex vishnui* mosquitoes. Indian J. Med. Res. 84: 283-285.

Southgate, V.R. and M.C. Agrawal. 1990. Schistosomiasis in India? Parasitol. Today 6: 165-168.

Srivastava, P.K., A.V. Sadanand, N.B.L. Saxena, R.K. Dasgupta and M.V.V.L. Narasimham. 1994. Impact of residual insecticidal spray on the incidence of Japanese encephalitis. J. Commun. Dis. 26: 59-60.

Stanton, B., S. Khanam, H. Nazrul, S. Nurani and T. Khair. 1987. Scabies in urban Bangladesh. J. Trop. Med. Hyg. 90: 219-226.

Tiwari, S.N., A. Prakash and S.K. Ghosh. 1997. Seasonality of indoor resting anophelines in a stone quarry area of District Allahabad, U.P. Indian J. Malariol. 34: 132-139.

Uppal, S.S., V. Agnihotri, S. Ganguly, S. Badhwar and K.J. Shetty. 1990. Clinical aspects of centipede bite in the Andamans. J. Assoc. Physicians India 38:163-164.

Varma, R and B. Mahadevan. 1973. The bionomics and vector potential of the scrub typhus vector, *Leptotrombidium deliense,* and other trombiculid populations in eastern Himalayas, India. Indian J. Med. Sci. 27: 900-919.

Vasanthathilaka, V.W. J.K. and N. Senanayake. 1995. Typhus fever from the mountainous areas in Central Sri Lanka. Kandy Med. J. 4: 6-9.

Webster, J.P., G. Lloyd and D.W. Macdonald. 1995. Q fever (*Coxiella burnetii*) reservoir in wild brown rat (*Rattus norvegicus*) populations in the UK. Parasitol. 110: 31-35.

Wickramasinghe, R.H., R.S.B. Wickramasinghe and C.L. Mendis. Some recent mosquito-related studies and development in Sri Lanka. Biomed. Environ. Sci. 7: 266-277.

Wolstenholme, R.J. 1984. The disease spectrum in a Maldivian (Adduan) population. Trans. R. Soc. Trop. Med. Hyg. 78: 505-507.

World Health Organization. 1989. Geographical distribution of arthropod-borne diseases and their principal vectors. WHO/VBC/89.967.

World Health Organizaton. 1996. Operational manual on the application of insecticides for the control of mosquito vectors of malaria and other diseases. WHO/CTD/VBC/96.1000. (available from DPMIAC)

Yadav, M.P., J.R. Rarotra and M.S. Sethi. 1979. The occurrence of coxiellosis among rodents and shrews in the Tarai area of Uttar Pradesh. J. Wildl. Dis. 15: 11-14.

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ae. aegypti		+		+	+		+	
Ae. albolateralis		+		+		+		+
Ae. alboniveus				+				
Ae. albopictus		+		+	+	+	+	+
Ae. alboscutellatus				+				
Ae. albotaeniatus				+		+		+
Ae. andamanensis		+		+				
Ae. annandalei		+		+				
Ae. annulirostris				+		+		+
Ae. assamensis		+		+		+		
Ae. barraudi				+				
Ae. butleri				+				+
Ae. cacharanus				+		+		
Ae. caecus		+		+		+		
Ae. caspius	+						+	
Ae. chrysolineatus		+		+		+		+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ae. cogilli				+		+		
Ae. craggi				+				
Ae. culicinus				+			+	
Ae. deccanus				+		+		
Ae. dissimilis		+		+		+		
Ae. elsiae				+	+		+	
Ae. feegradei							+	
Ae. formosensis		+		+	+		+	
Ae. gouldi							+	+
Ae. greeni				+		+		+
Ae. gubernatoris				+			+	+
Ae. harveyi				+			+	+
Ae. imprimens		+						
Ae. indicus				+			+	+
Ae. iyengari		+		+				
Ae. jamesi				+				+
Ae. khazani		+		+		+		

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ae. krombeini								+
Ae. lankaensis								+
Ae. lineatopennis		+		+		+	+	+
Ae. lophoventralis		+		+		+		
Ae. macdougalli				+		+		+
Ae. mediopunctatus				+				+
Ae. menoni				+				
Ae. micropterus				+			+	
Ae. niveoides				+		+		
Ae. niveus		+		+				+
Ae. novalbopictus				+				+
Ae. novoniveus				+				
Ae. nummatus				+				
Ae. ostentaio				+				+
Ae. pallidostriatus		+				+		+
Ae. pampangensis				+		+		
Ae. patriciae				+				

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ae. periskelatus				+				+
Ae. petroelephantus				+				+
Ae. pipersalatus		+		+		+	+	+
Ae. poicilus				+				
Ae. pseudoalbopictus				+		+		
Ae. pseudomediofasciatus				+				+
Ae. pseudotaeniatus		+		+		+		+
Ae. pulchribarsis	+			+				
Ae. pulchriventer				+		+		
Ae. pulverulentus							+	
Ae. punctifemoris		+		+		+	+	
Ae. ramachandrai				+				+
Ae. reginae		+		+				+
Ae. scatophagoides		+		+		+	+	+
Ae. seculatus								+
Ae. shortti				+			+	
Ae. simplex								+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ae. spermathecus								+
Ae. srilankensis								+
Ae. subalbopictus				+		+		
Ae. taeniorhynchoides				+			+	+
Ae. thomsoni		+		+		+	+	+
Ae. unilineatus				+		+	+	
Ae. vallistris				+				
Ae. vexans	+	+	+	+			+	+
Ae. vittatus		+		+		+	+	+
Ae. w-albus		+				+	+	+
Ae. yerburyi								+
Ae. yusafi				+				+
Aediomyia catasticta		+		+				+
Anopheles aconitus		+		+		+		+
An. ahomi		**?**		+		+		
An. aitkenii		+		+		+		+
An. algeriensis	+							

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
An. annandalei				+		+		
An. annularis	+	+		+		+	+	+
An. argyropus		**?**		+			+	+
An. barbirostris		+		+		+	+	+
An. barianensis				+			+	
An. barumbrosus		+		+		+		+
An. bengalensis		+		+		+	+	
An. claviger	+						+	+
An. culicifacies	+	+		+		+	+	+
An. culiciformis					+			
An. dirus		+			+		+	
An. dravidicus				+			+	
An. dthali	+				+		+	+
An. elegans				+				+
An. filipinae						+	+	
An. fluviatilis	+	+		+	+	+	+	+
An. gigas simlensis		+		+	+		+	+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
An. hyrcanus	+	+		+		+	+	+
An. indefinitus				**?**		+		
An. insulaeflorum		**?**		+				+
An. interruptus		**?**		+		+		+
An. jamesii		+		+		+		+
An. jeyporiensis	+	+		+		+		
An. karwari		+		+		+		+
An. kochi		+		+		+		
An. lindesayi	+	**?**		+		+	+	
An. maculatus	+	+		+		+	+	+
An. majidi		+		+		+		
An. minimus	+	+		+		+	+	+
An. multicolor	+			+			+	
An. nigerrimus		+		+		+	+	+
An. nitidus		**?**		+				
An. nivipes						+		
An. pallidus		+		+		+	+	+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
An. peditaeniatus	+	+	+	+		+		+
An. peytoni			+					+
An. philippinensis		+		+		+		
An. pinjaurensis				+				
An. pseudojamesii		+		+		+		+
An. pseudopictus	+							
An. pseudowillmori				+		+		
An. pulcherrimus	+			+			+	
An. reidi			+					+
An. roperi		**?**		+				
An. sacharovi	+							
An. sergentii							+	
An. sinensis		**?**		+		+		
An. sintoni				+				
An. splendidus		+		+		+	+	
An. stephensi	+	+		+		+	+	
An. subpictus	+	+	+	+	+	+	+	+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
An. sundaicus		+		+				
An. superpictus	+			**?**			+	+
An. tesselatus		+		+	+	+	+	
An. theobaldi		+		+	+		+	+
An. turkhudi	+			+	+		+	+
An umbrosus		+		+	+			
An. vagus	+	+		+	+		+	**?**
An. varuna		+	+	+	+	+	+	+
An. willmorei	+	+			+	+	+	
Armigeres annulitarsis		+		+	+			
Ar. aureolineatus				+			+	+
Ar. dentatus		+		+	+		+	
Ar. digitatus		+		+	+			
Ar. dolichocephalus					+	+	+	
Ar. durhami				+	+		+	
Ar. flavus	+	+		+				
Ar. inchoatus		+		+				

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ar. kesseli						+		
Ar. kuchingensis		+		+		+		
Ar. magnus		+		+		+		
Ar. omissus		+		+				
Ar. subalbatus		+		+		+		+
Cx. bailyi								+
Cx. barraudi				+		+		
Cx. bengalensis				+				
Cx. bicornutus								+
Cx. bitaeniorhynchus		+		+		+	+	+
Cx. brevipalpis		+		+	+	+		+
Cx. cinctellus				+				
Cx. deserticola	+							
Cx. edwardsi				+		+		
Cx. epidesmus		+		+		+	+	?
Cx. foliatus						+		
Cx. fragilis								+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Cx. fuscanus		+		+		+	+	
Cx. fuscocephala		+		+			+	+
Cx. gelidus		+		+		+		+
Cx. halifaxii		+		+		+	+	+
Cx. hortensis	+			+				
Cx. hutchinsoni		+		+		+		+
Cx. infantulus				+	+	+	+	+
Cx. infula				+	+	+	+	+
Cx. jacksoni						+		+
Cx. malayi		+		+	+	+	+	+
Cx. mimeticus				+		+		
Cx. mimulus		+		+		+		+
Cx. minor				+				
Cx. minutissimus		+	+		+			+
Cx. modestus	+							
Cx. nigropunctatus		+		+		+		+
Cx. pallidothorax		+		+		+	+	+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Cx. perexiguus	+			+			+	
Cx. perplexus				+				
Cx. pipiens	+				+			+
Cx. pluvialis				+				+
Cx. pseudovishnui		+		+		+	+	+
Cx. pusillus	+							
Cx. quinquefasciatus	+	+		+		+	+	+
Cx. rubithoracis				+				+
Cx. sinensis		+		+		+		+
Cx. sitiens		+		+	+			+
Cx. spathifurca					+			+
Cx. theileri	+	+				+	+	
Cx. tritaeniorhynchus		+		+	+	+	+	+
Cx. vagans		+				+	+	
Cx. viridiventer		+		+		+		
Cx. vishnui		+		+	+		+	+
Cx. wardi								+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
Cx. whitei		+		+		+		
Cx. whitmorei		+		+		+	+	+
Coquillettidia crassipes		+		+		+	+	+
Cq. novochracea				+				
Cq. ochracea		+						
Cq. richardii	+							
Culiseta alaskaensis indica				+			+	
Cs. longiareolata	+			+			+	
Cs. niveitaeniata				+		+	+	
Cs. subochrea	+							
Ficalbia minima		+		+				+
Heizmannia aureochaeta				+				
Hz. complex				+				
Hz. covelli		+		+				
Hz. greenii			+					+
Hz. himalayensis				+		+		
Hz. indica						+		

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
Hz. rerdi				+		+		
Malaya genurostris		+		+	+	+		+
M. jacobsoni		+		+		+		
Mansonia annulifera		+		+		+		+
Ma. dives		+		+				
Ma. indiana		+		+		+		+
Ma. uniformis		+		+		+		+
Mimomyia chamberlani		+		+		+	+	+
Mi. hybrida		+		+		+		+
Mi. luzonensis				+		+		+
Ochlerotatus portonovoensis				+				
Orthopodomyia albipes				+				
Or. anopheloides		+		+		+	+	+
Topomyia aureoventer				+		+		
Tp. ceylonensis								+
Toxorhynchites bengalensis		+						
Tx. splendens				+		+		+

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
Tripteroides aranoides		+		+				
Tr. indicus				+		+		+
Uranotaenia annandalei				+		+		
Ur. bicolor								+
Ur. campestris		+		+		+		+
Ur. edwardsi				+		+		
Ur. luteola				+		+		
Ur. macfarlanei				+		+		
Ur. maculipleura						+		
Ur. novobscura		+		+				
Ur. recondita				+		+		+
Ur. srilankensis								+
Ur. stricklandi				+		+		
Ur. unguiculata	+						+	
Verrallina abdita				+				
Ve. agrestis				+				
Ve. clavata				+				

Appendix A.1. Distribution of Mosquitoes in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Ve. comata				+				
Ve. pseudodiura				+				
Ve. rami				+				
Ve. uniformis								+

Appendix A.2. Distribution of Sand Flies in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Phlebotomus alexandri	+			+			+	
P. andrejevi	+							
P. angustus	+						+	
P. argentipes		+	?	+		+	+	+
P. burneyi							+	
P. caucasicus	+						?	
P. caudatus	+							
P. chinensis	+			+			+	
P. colabaensis				+		?	+	
P. comatus	+					+		
P. eleanorae				+				
P. hindustanicus	+			+		+	+	
P. kabulensis	+							
P. kandelakii	+			+			+	
P. kazeruni	+							
P. keshishiani	+						+	
P. longiductus	+			?		?	+	
P. major s. l.	+			+		+	+	
P. mascittii				+				
P. mesghalii	+						+	
P. mongolensis	+							
P. newsteadi				+				
P. nuri	+			+			+	
P. papatasi	+	+	?	+		+	+	?
P. pholetor								?
P. rupester	+						+	
P. salangensis	+							
P. salehi	+			+			+	

Appendix A.2. Distribution of Sand Flies in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
P. sanctijohani				+				
P. sergenti	+			+		+	+	
P. stantoni				+		+		+
P. teshi				+		+		
P. tubifer				+				
P. turanicus	+							
Sergentomyia africana magna		+					+	
S. arboris				+		+		+
S. babu	+	+		+		+	+	
S. baghdadis	+	+		+			+	
S. bailyi	+			+		+	+	
S. barraudi		+	+	?				
S. chakravarti				+				
S. cherukara				+				
S. christophersi				+			+	
S. clydei	+			+			+	
S. dentata	+						+	
S. dhandai				+				
S. eadithae				+		+		
S. fallax	+							
S. geoffrey						+		
S. grekovi	+							
S. himalayensis			+	+		+		
S. hodgsoni	+			+			+	
S. hospittii	+			+			+	
S. indica	+	+		+		+	+	
S. insularis				+				+
S. iyengari				+				
S. kauli						+		
S. kottamala				+				

Appendix A.2. Distribution of Sand Flies in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
S. kauli				+				
S. kurandamallai				+				
S. linearis				+				
S. malabarica		+		+				
S. mervynae	+							
S. modii				+				
S. montana	+			+		+	+	
S. palestinensis	+						+	
S. pawlowskyi	+						+	
S. perturbans		+	+	+		+		
S. punjabensis	+	+		+			+	+
S. purii		+	+	+		+		
S. shettyi				+				
S. shortii		+	+	+		+	+	
S. sintoni	+							
S. sirohi				+			+	
S. sogdiana	+							
S. squamipleuris	+							
S. sumbarica	+							
S. theodori s. l.	+	+		+			+	
S. tiberiadis pakistanica	+						+	
S. vergehesei				+		+		
S. zeylanica		+	+	+		+		+

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
ARGASIDAE								
Argas abdussalami	+			+			+	
A. boueti	+	?	?	?		?	?	
A. ceylonensis								+
A. confusus	+							
A. hermanni	+			+		+	+	
A. himalayensis						+		
A. persicus	+		?	+		+	+	
A. pusillus		+						
A. reflexus	+							
A. robertsi			?	+				+
A. transgariepinus	+							
A. vespertilionis	+			+		+	+	
Ornithodoros chiropterphila				+				
O. coniceps	+			+		+	?	+
O. indica				+				
O. lahorensis	+			+			+	

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
O. moubata			**?**	**?**		**?**		
O. papillipes	+							
O. piriformis				+		+		
O. savignyi				+				
O. tartakovskyi	+						**?**	
O. tholozani	+			+			+	
Otobius megnini				+				
IXODIDAE								
Amblyomma clypeolatum				+				+
A. geoemydae		**?**		+				
A. integrum			+	+		+		+
A. javanense				+			+	
A. nitidum				+				
A. supinoi				+				
A. testudinarium		+	**?**	+		+		+
Anomalohimalaya lama				**?**		+		
Aponomma gervaisi	+	+		+			+	+
A. pattoni				+				+

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
A. varanensis		?		+				+
Boophilus annulatus	+	+	?	+		?	+	+
B. microplus	+	+	+	+		+	+	+
Dermacentor atrosignatus						+		
D. auratus	+	+	+	+		+	+	+
D. everestianus						+		
D. marginatus	+						+	
D. niveus	+						+	
D. raskemensis	+			+			+	
Haemaphysalis aborensis				+				
H. aculeata				+				+
H. anomala	+		?	+		+	+	+
H. aponommoides	+		?	+		+	+	
H. birmaniae				+		+		
H. bispinosa	+	+	+	+		+	+	+
H. campanulata				+				
H. canestrinii		+		+		+		
H. cornigera			?	+		+		?

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. cornupunctata	+		?	+		?	+	
H. cuspidata				+				+
H. danieli	+			+		+	+	
H. darjeeling				+				
H. davisi			?	+		?		
H. doenitzi				+		+		
H. erinacei	+							
H. e. turanica	+							
H. flava				+				
H. garhwalensis	+		?	+		+	+	
H. howletti				+		+	+	
H. hystricis	+	+	?	+		+	+	+
H. indica	+			+		+	?	+
H. indoflava				+				
H. intermedia			+	+			+	+
H. kashmirensis	+			+			?	
H. kinneari		+		+				
H. kopetdaghica	+			+		+	+	

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. kutchensis				+			+	
H. kyasanurensis				+				+
H. longicornis				+				
H. megalaimae				+				
H. minuta				+		+		?
H. montgomeryi	+		?	+		+	+	
H. nepalensis	+		?	+		+	+	
H. obesa				+				
H. ornithophila		?	?	?		+		
H. papuana			?	+		?		
H. paraturturis				+				
H. pospelovashtromae	+			+		+	+	
H. punctata	?			?			?	
H. ramachandrai			?	+		+		
H. sambar				+				
H. shimoga				+				
H. silvafelis				+				
H. spinigera	+		?	+		+	+	+

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. sulcata	+			+			+	
H. sundrai			?	+		+	+	
H. tibetensis	+			+		+	+	
H. turturis				+				+
H. warburtoni	+		?	+		+	+	
H. wellingtoni				+		+		
Hyalomma aegyptium	+			+			+	
H. a. anatolicum	+	+	?	+		+	+	
H. a. excavatum	+			+			+	
H. asiaticum	+						+	
H. brevipunctata				+		+	+	
H. detritum	+	?	?	+		+	+	
H. dromedarii	+			+			+	
H. hussaini				+			+	
H. hystricis				+				
H. impeltatum	+						+	
H. kumari	+			+		+	+	+
H. marginatum marginatum	+		?	+		+	+	+

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. m. isaaci	+		?	+		+	+	+
H. m. turanicum	+	?	?	+		+	+	
H. schulzei	+						+	
H. truncatum		+						
Ixodes acuminatus	+							
I. acutitarsus	+		+	+		+	+	
I. arboricola	+							
I. berlesei						+		
I. ceylonensis				+				+
I. crenulatus	+			+			+	
I. granulatus	+		?	+		+	+	
I. himalayensis	+			+		+	?	
I. hyatti			?	?		+	+	
I. kashmiricus	+		?	+		+	+	
I. kuntzi						+		
I. mitchelli			?	?		+		
I. moschiferi						+		
I. nuttallianus			?	?		+		

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
I. ovatus	+		?	+		+	+	
I. petauristae				+				+
I. redikorzevi	+					+	+	
I. shahi			?	?		+	+	
I. simplex	+							
I. stromi							+	
I. tanuki						+		
I. turdus						+		
I. verspertilionis	+							
Nosomma monstrosum	+		?	+		+	+	+
Rhipicephalus arakeri				+				
R. bursa	?			?				
R. e. evertsi	?	+		?			?	
R. haemaphysaloides	+		?	+		+	+	+
R. leporis	+							
R. pumilio	?							
R. ramachandrai				+			+	?
R. sanguineus	+	+	?	+		+	+	+

Appendix A.3. Distribution of Ticks in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
R. scalpturatus				+		+		
R. turanicus	+	?	?	+		+	+	

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
ANCISTROPSYLLIDAE								
Ancistropsylla nepalensis		+	+	+		+		
CERATOPHYLLIDAE								
Amphalius c. clarus			+			+		
A. r. runnatus	+							
Callopsylla c. caspia	+							
C. c. fragilis	+							
C. c. gaiskii	+							
C. c. tiflovi	+							
C. changduensis	+							
C. dolabella			+			+		
C. dolabris	+							
C. kozlovi	+							
C. lagomys	+							
C. mygala			+			+		
C. oreinus							+	

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
C. saxatilis	+							
C. sparsilis atallahi			+			+		
C. streeti	+							
Ceratophyllus caliotes							+	
C. fringillae	+							
C. hirundinis	+						+	
C. orites							+	
C. phrillinae			+			+		
C. v. vagabundus	+							
Citellophilus l. lebedewi	+							
C. trispinus	+							
Macrostylophora fimbriata		+		+				
M. hastata nepali			+			+		
M. h. sikkimensis		+		+				
M. l .lupata			+			+		
M. phillipsi								+

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Megabothris rhipisoides			+			+		
Monopsyllus sciurorum asiaticus	+							
Nosopsyllus afghanus	+							
N. alladinis		+		+				
N. arcotus		+		+				
N. argutus		+		+				
N. ceylonensis								+
N. eremicus	+							
N. fasciatus		+		+				
N. fidus	+							
N. nilgiriensis		+		+				
N. punensis		+		+				
N. punjabensis	+	+	+	+		+		
N. simla	+	+	+	+		+	+	
N. tamilanus								+
N. t. turkmenicus	+							
N. t. altisetus	+							

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
N. vauceli		+		+				
N. vlasovi	+							
Oropsylla silantiewi	+							
Rowleyella arborea			+			+		
Smitipsylla maseri			+			+		
S. prodigiosa		+		+				
COPTOPSYLLIDAE								
Coptopsylla afghana	+							
C. barbarae	+							
C. janiceae	+							
C. l. lamellifer	+							
C. o. olgae	+							
CTENOPHTHALMIDAE								
Doratopsylla coreana araea			+			+		
D. wissemani							+	
Genoneopsylla longisetosa			+			+		
Neopsylla angustimanubra			+			+		
N. d. dispar		+		+				

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
N. heckeli							+	
N. hissarica	+							
N. mantissa			+			+		
N. marleanae			+			+		
N. pagea								
N. pleskei ariana	+							
N. s. secura			+			+	+	
N. s. kashmirensis		+		+				
N. s. separta			+			+		
N. s. setosa	+							
N. stevensi stevensi			+			+		
Palaeopsylla aporema			+			+		
P. daniela			+			+		
P. helenae			+			+		
P. remota		+		+				
P. setzeri							+	
P. t. tauberi			+			+		
P. t. makaluensis			+			+		

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
P. ioffi nepali			+			+		
Rhadinopsylla alticola							+	
R. dahurica vicinoides			+			+		
R. kullmanni	+							
R. li murium	+							
R. rhigalea			+			+		
Stenischia lewisi			+			+		
S. pagiana			+			+		
S. rhadinopsylloides			+			+		
Stenoponia himalayana			+			+		
Stenischia himalayana			+			+		
Xenodaeria telios			+			+	+	
HYSTRICHOPSYLLIDAE								
Hystrichopsylla synaptica			+			+		
ISCHNOPSYLLIDAE								
Araeopsylla gestroi								+
Chiropteropsylla brockmani	+							
Ischnopsyllus delectabilis		+		+				

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
I. indicus		+	+	+		+	+	+
I. obscurus	+							
I. octactenus	+							
Lagaropsylla putilla		+		+				
Mitchella exsula			+			+		
Rhinolophopsylla u. unipectinata	+							
R. u. indica		+		+				
R. u. turkestanica	+							
Thaumapsylla breviceps		+		+			+	
LEPTOPSYLLIDAE								
Aconothobius anthobius			+			+		
A. martensi			+			+		
A. orientalis		+	+	+		+		
Acropsylla episema		+		+				
A. traubi	+							
Amphipsylla anceps	+							
A. kulkarnii		+	+	+		+	+	
A. montana	+	+	+	+		+	+	

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
A. montium	+							
A. orthogonia		+	+	+		+	+	
A. parthiana	+							
A. phaiomydis limonia	+	+	+	+		+	+	
A. p. primaris		+	+	+		+	+	
A. quadratedigita		+	+	+		+	+	
A. rossica	+							
A. schelkovnikovi	+							
A. tuta gregorii		+	+	+		+	+	
Caenopsylla l. laptevi	+							
Frontopsylla ambigua	+						+	
F. cornuta			+			+		
F. diqiengensis			+			+		
F. elata vara		+		+			+	
F. e. elatoides	+							
F. frontalis alatau	+							
F. mutada	+							
F. ornata	+							

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
F. spadix nepalensis			+			+		
Geusibia lacertosa			+			+		
G. triangularis			+			+		
Leptopsylla nana	+							
L. pamirensis	+						+	
L. segnis		+	+	+		+		+
L. sexdentata	+							
Mesopsylla eucta afghana	+							
Ochotonobius rufescens	+							
Ophthalmopsylla v. volgensis	+							
O. celata							+	
Paradoxopsyllus acanthus			+			+		
P. custodis			+			+		
P. dictosus			+			+		
P. digitatus			+			+		
P. hollandi			+			+		
P. magnificus			+			+		
P. microphthalmus	+							

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
P. mustangensis			+			+		
P. oribatus			+			+		
P. paraphaeopis			+			+		
P. spinosus			+			+		
Peromyscopsylla h. himalaica		+	+	+		+		
Phaenopsylla hopkinsi	+							
P. jordani	+							
P. mustersi	+							
PULICIDAE								
Ctenocephalides canis	+	+	+	+	+	+	+	+
C. f. felis	+	+	+	+	+	+	+	+
C. orientis	+	+	+	+	+	+	+	+
C. paradoxuri								+
E. gallinacea	+	+		+				+
E. liopus		+		+				
Euhoplopsyllus glacialus profugus	+							
Pariodontis riggenbachi wernecki		+		+				
Pulex irritans	+	+	+	+	+	+	+	+

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
Synosternus c. cleopatrae	+							
S. longispinus	+							
S. pallidus	+	+		+				
Tunga pentrans		+		+			+	
Xenopsylla astia	+	+	+	+	+	+	+	+
X. brasilensis		+		+			+	+
X. c. cheopis		+	+	+	+	+	+	+
X. conformis conformis	+							
X. g. gerbilli	+							
X. hussaini		+		+				
X. magdalinae	+							
X. nesokiae	+							
X. nubica	+							
X. persica	+							
PYGIOPSYLLIDAE								
Lentistivalius ferinus		+	+	+		+		+
Stivalius a. aporus		+	+	+		+		
S. phoberus								+

Appendix A.4. Distribution of Fleas in South Central Asia (+ = Present; ? = Uncertain)								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
VERMIPSYLLIDAE								
Chaetopsylla globiceps	+							
C. gracilis			+			+		
C. homoea		+		+				
Vermipsylla p. perplexa			+			+		

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
BOTHRUIDAE								
Cercophonius himalayensis				+				
BUTHIDAE								
Afganorthochirus danielliae	+							
A. erardi	+						**?**	
A. monodi	+							
Androctonus amoreuxi baluchicus	+						+	
A. finitimus				+			+	
Baloorthochirus becvari							+	
Buthoscorpio politus				+				
B. sarasinorum				+				+
Charmus indicus				+				
C. laneus								+
C. sinhagadensis				+				
Compsobuthus rugosulus	**?**			+				+
Hemibuthus crassimanus				+				
Himalayotityobuthus martensi				+		**?**	+	
Hottentotta a. alticola	+						+	
H. a. kabulensis	+							
H. a. minusalta	+							
H. a. nigrifrons							+	

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. a. penjabensis				+			+	
H. kraepelini				+				
Isomertus (Isomertus) isadensis				+				
I. (I.) maculatus				+	+		+	+
I. (I.) sankeriensis				+				
I. (I.) thurstoni				+				+
I. (I.) thwaitesi								+
I. (Reddyanus) a. acanthurus				+				
I. (R.) a. loebli								+
I. (R.) assamensis				+				
I. (R.) basilicus				+				
I. (R.) besucheti								+
I. (R.) brachycentrus				+				
I. (R.) corbeti				+				
I. (R.) rigidulus				+				
I. (R.) vittatus				+				
Lychas biharensis				+				
L. farkasi						+		
L. hendersoni				+				
L. heurtaultae						+		
L. hillyardi				+				

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
L. kamshetensis				+				
L. kharpadi				+				
L. laevifrons				+		+		
L. mucronatus				+				
L. nigristernis				+		+		
L. rackae				+				
L. rugosus				+				
L. scaber				+				
L. srilankensis								+
L. tricarinatus				+				
Mesobuthus eupeus afghanus	+							
M. e. haarlovi	+							
M. e. pachysoma							+	
M. e. persicus							+	
M. hendersoni				+				
M. macmahoni	+						+	
M. pachyurus								
M. rugiscutis				+				
M. t. tamulus				+				
M. t. concanesis				+				
M. t. gangeticus				+				
M. t. gujaratensis				+				
M. t. sindicus				+			+	

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
Odontobuthus ondonturus				+			+	
Oliverus caucasicus parthorum	+							
Orthochirus bicolor	+			+			+	
O. flavescens				+				
O. fuscipes							+	
O. krishnai				+				
O. lutipes				+				
O. pallidus	+			+			+	
O. scrobiculosus	+			+			+	
O. s. melanurus				**?**				
Pakistanorthocirus weitschati							+	
Paraorthochirus blandini							+	
Plesiobuthus paradoxis							+	
Sassanidothus zarudnyi gracilis	+						+	
CHAERILIDAE								
Chaerilus a. anthracinus				+				
C. a. rufescens				+				
C. assamensis				+				
C. ceylonensis								+
C. gemmifer		+						
C. granifrons				**?**				

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
C. granosus				+				
C. hirsti				+			+	
C. insignis				+				
C. pictus		+		+				
C. tricostatus				+				
C. truncatus				+				
Vachonus atrostriatus				+			+	
V. rajasthanicus				+			**?**	
ISCHNURIDAE								
Chiromachetes fergusoni				+				
C. tirupati				+				
Lomachus l. laeviceps				+				
I. l. malabarensis				+				
I. nitidus				+				
I. punctulatus				+				
I. surgani				+				
Liocheles australasiae				+				
L. nigripes				+				
SCORPIONIDAE								
Hemiscorpius lepturus							+	
Heterometrus leioderma				+				

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
H.(Chersonesometrus) barberi				+				
H. (C.) collinus				+				
H. (C.) fastigiosus				+				
H. (C.) fulvipes				+				
H. (C.) granulomanus				+				
H. (C.) karanensis				+				
H. (C.) linuris				+				
H. (C.) madraspatensis				+				
H. (C.) pelekomanus				+				
H. (C.)phipsoni				+				
H. (C.) s. obscurus				+				
H. (C.) s. scaber				+				
H. (C.) s. rugusus				+				
H. (C.) tristis				+				
H. (C.) wroughtoni				+				
H. (C.) xanthopus				+				
H. (Giganometrus) flavimanus				+				
H. (G.) s. swammerdami				+				
H. (G.) s. titanicus								+
H. (Heterometrus) bengalensis				+				
H. (H.) keralaensis				+				

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).

Species	Afghanistan	Bangladesh	Bhutan	India	Maldives	Nepal	Pakistan	Sri Lanka
H. (H.) malapuramensis				+				
H. (Srilankametrus) gravimanus				+				+
H. (S.) i. indus								+
H. (S.) i. laevitensus				+				
H. (S.) latimanus				+				
H. (S.) serratus								+
SCORPIOPIDAE								
Euscorpiops asthenurus				+				
E. bhutanensis			+					
E. lindbergi	+							
E. longimanus		+		+				
Neoscorpiops deccanensis				+				
N. satarensis				+				
N. tenuicauda				+				
Scorpiops affinis				+				
S. crassimanus				+				
S. hardwicki			+	+		+		
S. insculptus				+				
S. irenae						+		
S. kraepelini							+	

Appendix A.5. Distribution of Scorpions in South Central Asia (+ = Present; ? = Uncertain).								
Species	**Afghanistan**	**Bangladesh**	**Bhutan**	**India**	**Maldives**	**Nepal**	**Pakistan**	**Sri Lanka**
S. leptochirus		+		+				
S. montanus			+	+			+	
S. pachmarhicus				+				
S. p. petersii			+	+				
S. p. vonwicki				+				
S. rohtangensis				+			+	

Appendix B.1. Vector Ecology Profiles of Malaria Vectors in South Central Asia.				
Species	**Larval Habitats**	**Feeding Behavior**	**Resting Behavior**	**Flight Behavior**
An. aconitus	Rice fields, stream pools, shaded pools with grasses.	Feeds on man and animals, indoors and outdoors.	Indoors and outdoors.	Moderately strong flier, 1.5 to 2.5 km.
An. annularis	Rice fields, permanent water with emerging vegetation.	Generally zoophilic, feeding outdoors before midnight.	Outdoors.	Flies up to 1.7 km.
An. campestris	Brackish water, ditches, wells.	Primarily zoophilic, indoors or outdoors, bites in partial shade.	Indoors and outdoors.	Moderate range flier.
An. culicifacies	Irrigation canals, borrow pits, stream margins.	Feeds indoors, but more often outdoors. Prefers cattle to humans. Feeds at night up to 0300 hrs.	Mostly outdoors.	Flies up to 5.2 km.
An. dirus	Isolated stream pools, undisturbed ground pools, cisterns.	Highly anthropophilic, feeds late (2200 to 0400), indoors and outdoors.	Indoors and outdoors.	Flies 2 to 4 km.
An. fluviatilis	Stream beds, vegetated ponds, swamp edges.	Feeds on man and animals at night until 0300 hrs.	Indoors and outdoors.	Flight range of 0.5 to 1.5 km.
An. jeyporiensis	Rice fields, seepages, grassy streams, pond edges.	Man and animals, indoors and outdoors.	Indoors and outdoors.	Flies 800 m or more.
An. leucosphyrus	Isolated stream pools, ground pools, cisterns in shade.	Anthropophilic, feeds indoors and outdoors, 2200 to 0400 hrs.	Indoors and outdoors.	Flies up to 800 m.
An. maculatus	Stream margins, pond edges, ditches, rice fields.	Feeds on man and animals between 1800 and 2100 hrs.	Rests outdoors after feeding.	Flies up to 1 km.
An. minimus	Stream margins, ground pools, irrigation ditches, seepages.	Feeds strongly on man, but also on animals, with most feeding early in the evening.	Rests indoors.	Flies up to several km.
An. nigerrimus	Deep ponds, swamps, borrow pits.	Feeds on man and animals outdoors, early in the evening, or sometimes after dawn.	Rests outdoors.	Unknown flight range.
An. philippin-ensis	Rice fields, sunlit water with submerged vegetation.	Primarily zoophilic, feeding mostly outdoors.	Usually rests outdoors.	Short range flier, < 0.8 km.
An. pulcher-rimus	Stream margins and stream pools, rice fields, irrigation plots.	Prefers to feed outdoors on cattle, usually before midnight.	Rests indoors and outdoors.	Flies 5 km or more.
An. sergentii	Springs, date palm and rice irrigation plots.	Bites man and other animals, indoors and outdoors.	Rests in human dwellings or	Moderate flight range, may exceed 5 km.

Appendix B.1. Vector Ecology Profiles of Malaria Vectors in South Central Asia.				
Species	**Larval Habitats**	**Feeding Behavior**	**Resting Behavior**	**Flight Behavior**
			caves.	
An. subpictus	Muddy pools, rain gutters, brackish water.	Zoophilic, feeds indoors and outdoors, early in the evening.	Rests outdoors.	Flies up to 3.2 km.
An. superpictus	Clear, sunlit water, usually without vegetation.	Bites man and other animals, indoors and outdoors.	Rests in human dwellings, animal shelters, or caves.	Short to medium range flier; rarely flies >5 km from larval habitat.
An. sundaicus	Fish ponds, brackish water with algae present.	Prefers cattle and pigs to humans, feeds outdoors, often at high altitudes.	Rests outdoors.	Flies up to 5 km.
An. umbrosus	Stagnant water, ditches, pond edges.	Primarily zoophilic, outdoor feeder both early evening and after dawn.	Rests outdoors.	Unknown.
An. varuna	Stagnant water, ground pools, stream margins.	Bites man and animals indoors and outdoors.	Rests indoors.	Flies 1 km or less.

Appendix B.2. Vector Ecology Profiles of Common Tick Vectors in South Central Asia.				
Species	**Geographic Distribution**	**Potential Hosts**	**Disease Transmission**	**Bionomics/Habitat Information**
Boophilus microplus	India and Pakistan.	Adults and immatures feed on cattle, sheep, rarely horses and man.	A minor CCHF vector.	A 1-host tick. All stages of the life cycle are usually spent on cattle. Females rest up to a month before laying eggs, after feeding and mating. Life cycle takes <1 year.
Haemaphysalis spinigera	India and Pakistan.	Adults feed on deer and livestock, especially cattle. Immatures feed on hedgehogs, rabbits or birds.	Primary vector of Kyasanur Forest disease.	A 3-host tick. Larvae hatch in post-monsoon period from Oct to Dec. Larvae do not survive monsoon period. Nymphs active from Feb to May. Adults active June to Aug. Females lay 1,000 to 2,000 eggs.
H. turturis	India and Pakistan.	Adults prefer cattle but feed occasionally on humans and monkeys.	Vector of Kyasanur Forest disease.	Similar to *H. spinigera*, except life cycle lags 6 to 8 weeks behind.
H. wellingtoni	India and Pakistan.	All stages feed on ground-dwelling birds.	Vector of Kyasanur Forest disease.	Similar to *H. spinigera*.
Hyalomma a. anatolicum	Afghanistan, India and Pakistan.	Adults feed on monkeys, cattle, buffalo and humans. Immatures feed on porcupines, rats or birds.	Vector of CCHF. Transovarial transmission occurs.	A 3-host tick. Dispersed widely from steppes and deserts, often along caravan and cattle routes. Ticks common in feedlots. Nymphs feed on host's ears. Species often is active in winter. Aggressive host-seeker. Resists climatic extremes and aridity.
H. marginatum	Afghanistan, India, Nepal, Pakistan and Sri Lanka.	Adults feed on cattle, goats, and horses . Immatures feed on rabbits, hedgehogs, rats and birds.	Vector of CCHF. Transovarial transmission occurs.	A 2-host tick that survives well in cold, arid areas. Adults overwinter; eggs are laid in spring. Females feed 6 to 12 days. Immatures quest from rodent burrows. Life cycle takes 1 to 2 yrs.
Ixodes petauristae	India and Sri Lanka.	Adults prefer feeding on shrews, squirrels, rats, and cattle. Occasionally feed on humans and monkeys.	Kyasanur Forest disease. Transovarial transmission occurs in the lab.	Adults feed 8 to10 days. Nymphs feed 4 to 5 days. Females lay 1000 to 2000 eggs.

Appendix B.2. Vector Ecology Profiles of Common Tick Vectors in South Central Asia.

Species	Geographic Distribution	Potential Hosts	Disease Transmission	Bionomics/Habitat Information
Ornithodoros tholozani	Afghanistan, India and Pakistan.	Adults feed on camels, sheep, birds and rarely humans. Immatures feed on gerbils and other small mammals.	Primary vector of tick-borne relapsing fever.	Multi-host soft tick. Found in caves, huts, cabins or stables. Feeds in a few hours, usually at night. Usually has 3 to 4 immature instars. Females mate and may live several years without a bloodmeal. Often inhabits rodent burrows.
Rhipicephalus haemaphysaloides	India and Pakistan.	Feeds on domestic and wild animals.	Transmits Kyasanur Forest disease.	A 3-host tick. Life cycle occurs in 87 to 945 days. Copulation occurs on the host. Adults feed 7 to 9 days; larvae feed 1 to 5 days. Females lay 6,000 to 10,000 eggs.
R. sanguineus	Occurs throughout the region.	Adults and immatures feed on dogs, cattle, horse, sheep, and sometimes man.	Primary vector of boutonneuse fever.	A 3-host tick. Adults frequent the ears, or between toes of dogs. Immatures prefer long hair at the back of the neck. Females crawl upward and lay eggs in cracks of walls or ceilings of houses.

Appendix C. Pesticide Resistance in South Central Asia.

Vector-borne diseases are an increasing cause of death and suffering in many areas of the world. Efforts to control these diseases have been founded on the use of chemical pesticides. However, the spread of resistance among arthropods has rendered many pesticides ineffective, while few substitute pesticides are being developed. Resistance has been reported to every class of insecticides, including microbial agents and insect growth regulators.

Resistance is formally defined by the WHO as "the development of an ability in a strain of some organism to tolerate doses of a toxicant that would prove fatal to a majority of individuals in a normal population of the same species." Resistance has a genetic basis and is the result of a change in the genetic composition of a population as a direct result of the selection effects of the pesticide.

Early detection and monitoring are vital to resistance management. Historically, standardized methods, test kits and insecticides were provided by WHO. The simplest method of detecting resistance is the diagnostic dose test. The diagnostic dose is a predetermined insecticide dose known to be lethal to a high proportion of susceptible individuals, but that a high proportion of resistant individuals can tolerate. A list of recommended diagnostic doses of many insecticides for a number of arthropod vectors is available from WHO. For terrestrial and/or adult stages, the insecticide is either applied topically or insects are exposed to a surface treated with insecticide. For aquatic stages, insecticide is added to water at given concentrations.

New approaches use rapid biochemical tests to detect resistance and determine resistance mechanisms. These methods permit rapid multiple assays of a single specimen. Worldwide application of biochemical assays will require production of standardized kits similar to the insecticide bioassay kits supplied by WHO. The choice of method to test for resistance is of great importance in order to determine resistance mechanisms. Consult TG 189, Procedures for the Diagnostic Dose Resistance Test Kits for Mosquitoes, Body Lice, and Beetle Pests of Stored Products. To obtain test kits and additional recommendations for resistance testing contact:

USACHPPM/Entomology Science Programs
5158 Blackhawk Road
Aberdeen Proving Ground, MD 21010-5422
Tel: (410) 436-3613
DSN 584-3613, FAX (410) 436-2037

Pesticide resistance can be classified into 2 broad categories: physiological and behavioral. There are many mechanisms of physiological resistance, including reduced penetration of insecticides through the cuticle, presence of enzymes that detoxify the insecticide, and reduced sensitivity of the target site of the insecticide. Physiological resistance can confer cross-resistance to structurally related insecticides of the same chemical class or related classes. Some vector populations have acquired several resistance mechanisms providing multiple resistance to a variety of insecticide classes. Many vector control programs have reached a stage where resistance is so great that few chemical alternatives are available.

In recent years, synthetic pyrethroids have replaced widely used classes of insecticides such as organophosphates, carbamates, and chlorinated hydrocarbons. These pyrethroids have shown great promise for vector control due to their low mammalian toxicity and ability to quickly immobilize and kill arthropods at low dosages. Unfortunately, resistance has been detected in several medically important arthropods. An issue of concern in vector control is whether DDT resistance confers cross-resistance to pyrethroids as a result of similar resistance mechanisms. Increasing pyrethroid resistance is of particular concern to the US military because of the widespread use of permethrin and other pyrethroids in BDUs, bednets, and vector control programs. Studies indicate that resistance appears rapidly in areas where treated bednets are used to control mosquitoes.

Changes in behavior that result in reduced contact with an insecticide include a reduced tendency to enter treated areas or an increased tendency to move away from a surface treated with insecticide once contact is

made. These are population-based changes in a species' genetics resulting from the selection pressure of insecticide use. Avoidance behavior is widespread but poorly understood. Some form of behavioral avoidance has been documented for virtually every major vector species. Methods to detect and determine behavioral resistance have not been standardized and are difficult to interpret.

Pesticide resistance will be an increasing problem for vector control personnel. More than 90% of all pesticides are used for agricultural purposes. Insecticide resistance in at least 17 species of mosquitoes in various countries has occurred because of indirect selection pressure by agricultural pesticides. The agricultural use of insecticides in rice paddies has greatly contributed to the development of resistance in several species of *Anopheles* and *Culex* in many areas of South Central Asia.

Innumerable genetic, biologic and operational factors influence the development of insecticide resistance. A pesticide use strategy that will prevent the evolution of resistance has not been developed. Tactics to manage or delay the development of resistance include: 1) using nonchemical methods of control as much as possible, 2) varying the dose or frequency of pesticide application, 3) using local rather than area-wide application, 4) applying treatments locally only during outbreaks of vector-borne diseases, 5) using less persistent pesticides, 6) treating only certain life stages of the vector, 7) using mixtures of pesticides with different modes of action, 8) using improved pesticide formulations, 9) rotating pesticides having different modes of action, and 10) using synergists.

Reports of resistance must be interpreted carefully. Many reports of resistance for a vector species are based on single data sets from a single point within a country and may be years if not decades old. Resistant populations tend to revert to susceptible status once insecticide selection pressure has been removed. Isolated reports of resistance, although recent, may indicate local resistance that has not become widespread. Vector control personnel frequently assume that resistance in a particular species occurs throughout their control area but, in reality, insecticide resistance is focal. The length of time an insecticide has been used at a location may not be helpful in predicting the presence of resistance. Vectors in some countries have never developed resistance to DDT, despite decades of use in malaria control. Only appropriate resistance monitoring can guide the vector control specialist in the selection of a suitable insecticide.

Published Reports of Insecticide Resistance Testing in South Central Asia.*

Bang, Y.H. 1985. Implications in the control of malaria vectors with insecticides in tropical countries of South-east Asia region. Part I. Insecticide resistance. J. Commun. Dis. 17: 199-218.

Bang, Y.H. 1985. Implications in the control of malaria vectors with insecticides in tropical countries of South-east Asia region. Part II. Consequences of insecticide use. J. Commun. Dis. 17: 300-310.

Lines, J.D. 1988. Do agricultural insecticides select for insecticide resistance in mosquitoes? A look at the evidence. Parasitology Today 4: 17-20.

WHO. 1986. Resistance of vectors and reservoirs of disease to pesticides. Tenth report of the WHO expert committee on vector biology and control. WHO Tech. Rep. Ser. 737: 87 pp.

Bangladesh

Elias, M., M. Rahman, and J.M. Mizanur Rahman. 1985. The DDT susceptibility status of *Anopheles philippinensis* - a mosquito vector of malaria in Bangladesh. Bangladesh Med. Res. Council. Bull. 11: 1-7.

India

Amalraj, D.D., N. Sivagnaname and R. Srinivasan. 1999. Susceptibility of *Phlebotomus argentipes* and *P. papatasi* (Diptera: Psychodidae) to insecticides. J. Commun. Dis 31: 177-180.

Ansari, M.A., V.P. Sharma, C.P. Batra, R.K. Razdan and P.K. Mittal. 1986. Village scale trial of the impact of deltamethrin (K-othrine) spraying in areas with DDT and HCH resistant *Anopheles culicifacies*. Indian J. Malariol. 23:127-131.

Ansari, M.A., V.P. Sharma, R.K. Razdan and P.K. Mittal. 1990. Field evaluation of deltamethrin against resistant *Anopheles culicifacies* in District Ghaziabad (U.P.) India. Indian J. Malariol. 27: 1-13.

Bagherwal, R.K., R.S. Sisodia, A. Sharma, R.S. Dhanotiya, S.B. Ghosal and A. Sharma. 1995. *In vitro* studies on the susceptibility of the tick *Hyalomma anatolicum* to acaricides using F.A.O. test kit. Indian Vet. J. 72: 332-335.

Bansal, S.K. and K.V. Singh. 1995. Susceptibility status of two species of Japanese encephalitis vectors to insecticides in the Thar desert, District Bikaner (Rajasthan). Indian J. Med Res. 101: 190-192.

Bansal, S.K. and K.V. Singh. 1996. Insecticide susceptibility status of some anophelines in District Bikaner, Rajasthan. Indian J. Malariol. 33: 1-6.

Bansal, S.K. and K.V. Singh. 1996. Susceptibility status of *Phlebotomus papatasi* and *Sergentomyia punjabaensis* (Diptera: Psychodidae) to some insecticides in District Bikaner (Rajasthan). J. Commun. Dis. 28: 28-32.

Basak, B. and N. Tandon. 1995. Observations on susceptibility status of *Phlebotomus argentipes* to DDT in district South 24-Parganas, West Bengal. J. Commun. Dis. 27: 196-197.

Baskar, P and N.J. Shetty. 1992. Susceptibility status of *Anopheles stephensi* Liston to insecticides. J. Commun. Dis. 24: 188-190.

Bhattacharya, S. 1982. The present susceptibility status of *Armigeres subalbatus* to hydrochlorin and different organophosphorus insecticides. Bull. Zool. Survey India 4: 9-12.

Bhattacharya, D.R., R. Handique, A. Prakash, P. Dutta, J. Mahanta and V.K. Srivastava. 1996. Insecticide susceptibility status of potential vectors of Japanese encephalitis in Dibrugarh District, Assam. J. Commun. Dis. 28: 67-69.

Biswas, S., K. Kumar and K. Singh. 1988. The *Stegomyia* survey and susceptibility status of *Aedes aegypti* to insecticides in Calcutta seaport area. J. Commun. Dis. 20: 253-259.

Chakraborti, S., D.T. Mourya, M.D. Gokhale and K. Banerjee. 1993. Insecticide susceptibility status and enzyme profile of *Aedes albopictus* populations from different localities of Maharashtra State. Indian J. Med. Res. 97: 37-43.

Chakravorthy, B.C. and M. Kalanasundaram. 1992. Selection of permethrin resistance in the malaria vector *Anopheles stephensi*. Indian J. Malariol. 29: 161-165.

Chand, S.K. and R.S. Yadav. 1991. Insecticide susceptibility of mosquito vectors in Sundargarh District, Orissa. Indian J. Malariol. 28: 65-68.

Chandra, G., J. Bhattacharya and A.K. Hati. 1995. Susceptibility status of *Phlebotomus argentipes* to DDT, dieldrin and malathion in Hoogly, West Bengal. J. Commun. Dis. 27: 247-249.

Chitra, S. and M.K.K. Pillai. 1985. Role of esterases in organophosphorus and carbamate resistance in an Indian strain of *Anopheles stephensi* Liston. Indian J. Exp. Biol. 10: 576-584.

Choudhury, J.D. and P.R. Malhotra. 1982. Susceptibility of *Anopheles philippinensis* to DDT and dieldrin in Assam. Indian J. Malariol. 19: 145-146.

Das, M., S.P. Srivastava and J.S. Khambre. 1986. Susceptibility of DDT, dieldrin and malathion resistant *Anopheles culicifacies* populations to deltamethrin. J. Am. Mosq. Control Assoc. 2: 553-555.

Das, N.G., I. Baruah, P.R. Malhotra, S.C. Das and C.K. Krishnan. 1987. Susceptibility of some *Anopheles* mosquitoes to DDT and dieldrin in Tezpur (Assam). Indian J. Pub. Health 31: 221-224.

Das, P.K. and P.K. Rajagopalan. 1980. Insecticide resistance in *Culex pipiens fatigans* and its relevance to vector control. Indian J. Med. Res. 72: 500-507.

Das, S.C., D.R. Nath, M. Bhuyan, N.G. Das, I. Baruah and P.K. Talukdar. 1989. Studies on malaria and filariasis vectors in Kamorta and Great Nicobar Islands. Indian J. Malariol. 26: 153-157.

Das-Gupta, R.K., N.B.L. Saxena, R.D. Joshi and J.S. Rao. 1995. DDT resistance in *Phlebotomus papatasi* in Bihar. J. Commun. Dis. 27: 124.

Deobhankar, R.B. 1990. Magnitude of DDT resistance in *Anopheles culicifacies* in Maharashtra State. J. Commun. Dis. 22: 77.

Deobhankar, R.B. and N.D. Palkar. 1986. Stability of DDT resistance in *Anopheles culicifacies* in Maharashtra State (India). Pestology 10: 20-22.

Deobhankar, R.B. and N.D. Palkar. 1990. Susceptibility status of malaria vectors *Anopheles culicifacies* and *A. stephensi* (Diptera: Culicidae) to DDT, dieldrin and malathion. Entomon 15: 252-256.

Ganguly, S.S., K.K.D. Gupta and P.K. Dutta. 1994. Evaluation of the susceptibility status of *Culex quinquefasciatus* larvae to a few organophosphorus insecticides based on logistic regression analysis. Indian J. Pub. Health 38: 8-13.

Herath, P.R.J. and G. Davidson. 1981. Multiple resistance in *Anopheles culicifacies* Giles. Mosq. News 41:325-327.

Herath, P.R.J., S.J. Miles and G. Davidson. 1981. Fenitrothion (ONS 43) resistance in the taxon *Anopheles culicifaces*. J. Trop. Med. Hyg. 84: 87-88.

Jacob, P.G., G. Geevarghese and S.M. Kulkarni. 1995. Susceptibility of larvae of five species of mosquito vectors of Japanese encephalitis to insecticides from Kolar District, Karnataka. J. Commun. Dis. 27: 55-57.

Kaul, S., R.S. Sharma and B.K. Borgohain. 1994. DDT resistance in *Sergentomyia shorttii* (Diptera: Psychodidae) in Kamrup, Assam - first report in *Sergentomyia* genus. J. Commun. Dis. 26: 100-102.

Khamre, J.S. and M.B. Kaliwal. 1988. Mosquitoes of Daman. Indian J. Malariol. 25: 109-111.

Khan, S.A., P. Dutta, K. Narain, R. Handique and V.K. Srivastava. 1997. Studies on day-time resting habits of JE vector mosquitoes in upper Assam with a note on insecticide susceptibility status. J. Commun. Dis. 29: 367-370.

Krishnarao, P, V.K. Raina and T.K. Ghosh. 1989. Susceptibility status of *Culex quinquefasciatus* (Rajahmundry strain) to insecticides. J. Commun. Dis. 21: 145-147.

Kulkarni, S.M., G. Geevarghese and P.J. George. 1992. Susceptibility status of five species of Japanese encephalitis vectors to insecticides from Kolar District, Karnataka. Indian J. Med. Res. 95: 297-300.

Kulkarni, S.M. and P.S. Naik. 1991. Susceptibility studies on *Culex tritaeniorhynchus* Giles, 1901, to insecticides in the state of Goa. Indian J. Med. Res. 93: 179-181.

Kumar, K., S. Jamil-Ur-Rahman, S.K. Sharma, K.S. Gill, R. Katyal, R. Kaur, T.G.Thomas and K. Barua. 1997. Entomological and rodent surveillance in plague-suspected areas during September 1994 and thereafter. Jpn. J. Med. Sci. Biol. 50: 97-11.

Kumar, K., R. Katyal, K.S. Gill, S. Jamil-Ur-Rahman and S. Rahman. 1996. Prevalence of rat fleas in and around Delhi (India) area and their susceptibility status to insecticides. Jpn. J. Med. Sci. Biol. 49: 57-62.

Kumari, R., B.R. Thapar, R.K. Das-Gupta, S.M. Kaul, S. Lal, Roop-Kumari and Shiv-Lal. 1998. Susceptibility status of malaria vectors to insecticides in India. J. Commun. Dis. 30: 179-185.

Mahadev, P.V.M., M.A. Ilkal, D.T. Mourya, V.A.T. Desai and K. Banerjee. 1993. *Aedes aegypti* (L.) in Ahmedabad City, Gujarat: distribution, dengue detection and susceptibility to insecticides. J. Commun. Dis. 25: 169-183.

Mathur, K.K. and S.J. Rahman. 1983. Susceptibility of *Culex* (C*ulex*) *tritaeniorhynchus* Giles adults and larvae to insecticides in Delhi area. J. Commun. Dis. 15: 193-199.

Mehrotra, K.N. 1991. Current status of pesticide resistance in insect pests in India. J. Insect Sci. 4: 1-14.

Mehrotra, K.N. 1993. Pesticide resistance in India. Chem. Ind. 7: 399-403.

Mehrotra, K.N. 1994. Fifty years of DDT in India: a historical review. Pestic. Res. J. 6: 111-116.

Mourya, D.T., G. Geevarghese, M.D. Gokhale, P.S. Shetty, P. Kandasamy, K.V. Shantha, N.C. Appavoo, B.M. Dama and P.P. Doke. 1998. Present insecticide susceptibility status of *Xenopsylla cheopis* from Beed District, Maharashtra State, India. Entomon 23: 211-217.

Mourya, D.T., M.D. Gokhale, S. Chakaborti, P.V.M. Mahadev and K. Banerjee. 1993. Insecticide susceptibility status of certain populations of *Aedes aegypti* mosquitoes from rural areas of Maharashtra State. Indian J. Med. Res. 97: 87-91.

Mourya, D.T., M.D. Gokhale and A.C. Mishra. 1994. Biochemical basis of DDT resistance in *Aedes aegypti* populations from a dengue affected area in Shahjahanpur City. Indian J. Med. Res. 99: 212-215.

Mukhopadhyay, A.K., S. Chakraborty, P.K. Karmakar and P. Banerjee. 1996. Insecticidal susceptibility status of *Anopheles stephensi* (Liston) in selected areas of Calcutta (West Bengal). Indian J. Pub. Health 40: 130-133.

Mukhopadhyay, A.K., S. Chakraborty, A.K. Kureel and V.R. Shivraj. 1987. Resurgence of *Phlebotomus argentipes* and *Ph. papatasi* in parts of Bihar (India) after DDT spraying. Indian J. Med. Res. 85: 158-160.

Mukhopadhyay, A.K., A.K. Hati, S. Chakraborty and N.B.L. Saxena. 1996. The effect of DDT on *Phlebotomus* sandflies in kala-azar endemic foci in West Bengal. J. Commun. Dis. 28: 171-175.

Mukhopadhyay, A.K., N.B.L. Saxena and M.V. Narasimham. 1992. Susceptibility status of *Phlebotomus argentipes* to DDT in some kala-azar endemic regions of Bihar, India. WHO/CTD/VBC/92.995.

Mukhopadhyay, A.K., S.N. Sinha, R.L. Yadav and M.V.V.L. Narasimham. 1993. Susceptibility status of *Culex quinquefasciatus* in Patna to insecticides. Indian J. Pub. Health 37: 57-60.

Prakash, A., D.R. Bhattacharya, P.K. Mohapatra and J. Mahanta. 1996. Current susceptibility status of *Anopheles minimus* Theobald in Assam. J. Commun. Dis. 28: 143-145.

Prakash, A., D.R. Bhattacharya, P.K. Mohapatra and J. Mahanta. 1998. Insecticide susceptibility of *Anopheles dirus* in Assam. J. Commun. Dis. 30: 291-292.

Pushpalatha, N. and V.A. Vijayan. 1994. Adult population differences in response to six insecticides in *Culex fuscocephala* Theobald. Southeast Asian J. Trop. Med. Pub. Health 25: 532-535.

Pushpalatha, N. and V.A. Vijayan. 1998. Bioassay of some important insecticides against the adults of two populations of *Culex vishnui* Theobald prevalent in Mandya and Mysore, Karnataka State (India). J. Entomol. Res. 22: 293-297.

Pushpalatha, N. and V.A. Vijayan. 1999. Isoenzyme profiles in relation to ecological status in two Japanese encephalitis vectors, Culex vishnui and Culex fuscocephala (Diptera: Culicidae). Entomon 24: 297-306.

Raghavendra, K., S.K. Subbarao, M.K.K. Pillai and V.P. Sharma. 1998. Biochemical mechanisms of malathion resistance in Indian *Anopheles culicifacies* (Diptera: Culicidae) sibling species A, B, and C: microplate assays and synergistic studies. Ann. Entomol. Soc. Amer. 91: 834-839.

Raghavendra, K., S.K. Subbarao and V.P. Sharma. 1997. An investigation into the recent malaria outbreak in district Gurgagon, Haryana, India. Current Science 73: 766-770.

Raghavendra, K., K. Vasantha, S.K. Subbarao, M.K. Pillai and V.P. Sharma. 1991. Resistance in *Anopheles culicifacies* sibling species B and C to malathion in Andhra Pradesh and Gujarat States, India. J. Am. Mosq. Control Assoc. 7: 255- 259.

Raghavendra, K., K. Vasantha, S.K. Subbarao, M.K. Pillai and V.P. Sharma. 1992. Differential selection of malathion resistance in *Anopheles culicifacies* A and B (Diptera: Culicidae) in Haryana State, India. J. Med. Entomol. 29: 183-187.

Rao, D.R., T.R. Mani, R. Rajendran, A.S. Joseph, A. Gajanana and R. Reuben. 1995. Development of a high level of resistance to *Bacillus sphaericus* in a field population of *Culex quinquefasciatus* from Kochi, India. J. Am. Mosq. Control Assoc. 11: 1-5.

Renapurkar, D.M. 1981. A study of multiple insecticide resistance in the rat flea. Pesticides 15: 16-17.

Renapurkar, D.M. 1988. Distribution and susceptibility of *Xenopsylla cheopis* to DDT in Maharashtra State, India. Insect Sci. Appl. 9: 377-380.

Renapurkar, D.M. 1990. Distribution and insecticide resistance of the plague flea *Xenopsylla cheopis* in Maharashtra State, India. Med. Vet. Entomol. 4: 89-96.

Revanna, M.A., V.A. Vijayan and N. Ninge Gowda. 1991. Larval susceptibility of *Culex* (*Culex*) *gelidus* Theobald, against four organophosphorus compounds in Mysore city. J. Commun. Dis. 23: 202-203.

Revanna, M.A. and V.A. Vijayan. 1993. Insecticide susceptibility of *Culex gelidus* Theobald, a Japanese encephalitis vector, in Mysore. Ann. Entomol. 11: 67-70.

Roy, R.G., C.P. Vijayan and S. Pattanayak. 1980. Susceptibility of *Anopheles fluviatilis* in some areas of Karnataka, Kerala and Goa (1973-78). Indian J. Med. Res. 72: 654-658.

Saha, G.K., M. Chattopadhyay, G. Chandra and A.K. Hati. 1999. Susceptibility status of malaria vectors of India to commonly used insecticides - a review. Environ. Ecol. 17: 783-798.

Sahu, S.S., K. Gunasekaran, P. Jambulingam and P.K. Das. 1990. Susceptibility status of *Anopheles fluviatilis*, *A. annularis* and *A. culicifacies* to insecticides in Koraput District, Orissa. Indian J. Malariol. 27: 51-53.

Sahu, S.S. and K.P. Patra. 1995. A study on insecticide resistance in *Anopheles fluviatilis* and *Anopheles culicifacies* to HCH and DDT in the Malkangiri District of Orissa. Indian J. Malariol. 32: 112-118.

Shafi,S. and R. Ahmad. 1989. Development of tolerance in *Chrysomyia rufifacies* to organochlorine and carbamate compounds. J. Environ. Biol. 10: 111-117.

Sharma, R.S. 1990. Susceptibility status of malaria vectors *Anopheles culicifacies* and *Anopheles stephensi* (Diptera: Culicidae) to DDT, dieldrin and malathion. Entomon 15: 252-256.

Sharma, R.S. 1991. Susceptibility status of malaria vector *Anopheles culicifacies* (Diptera: Culicidae) and other anophelines to DDT and dieldrin in Himachal Pradesh State, India. Mosq.-borne Dis. Bull. 8: 6-10.

Sharma, R.S. 1995. Susceptibility status of malaria vector *Anopheles culicifacies* Giles (Diptera: Culicidae) to DDT, dieldrin and malathion in Punjab. J. Insect Sci. 8: 63-65.

Sharma, R.S. 1999. Susceptibility of the malaria vector *Anopheles culicifacies* (Diptera: Culicidae) to DDT, dieldrin, malathion, and lambda-cyhalothrin. J. Vector Ecol. 24: 187-190.

Sharma, R.S., S.N. Sharma and R.S. Sharma. 1995. Susceptibility level of malaria vector *Anopheles culicifacies* (Diptera: Culicidae) to malathion. Mosq.-borne Dis. Bull. 12: 10-12.

Sharma, R.S., S.N. Sharma and R.S. Sharma. 1996. Susceptibility of DDT, dieldrin, and the development of malathion resis tance in *Anopheles culicifacies* (Diptera: Culicidae) in Ambala District of Haryanna, India. J. Vector Ecol. 1: 14-16.

Sharma, S.N. 1993. Preliminary observations on the susceptibility of three anopheline species to insecticides in Bishnugarh, District Hazaribagh, Bihar. J. Commun. Dis. 25: 36-37.

Sharma, S.N. and K. Kumar. 1996. Entomological profile of some Japanese encephalitis prone districts of Haryana, India. Mosq.-borne Dis. Bull. 13: 14-17.

Sharma, V.P., R.K. Chandrahas, A.M. Ansari, P.K. Srivastava, R.K. Razdan and C.P. Batra.. 1986. Impact of DDT and HCH spraying on malaria transmission in villages with DDT and HCH resistant *Anopheles culicifacies*. Indian J. Malariol. 23: 27-38.

Sharma, V.P., H.C. Uprety, N. Nanda, V.K. Raina, S.K. Parida and V.K. Gupta. 1982. Impact of DDT spraying on malaria transmission in villages with resistant *Anopheles culicifaces*. Indian J. Malariol. 19: 5-12.

Singh, K.V. and S.K. Bansal. 1996. Insecticide susceptibility of *Phlebotomus papatasi* to organochlorine, organophosphate and carbamate compounds in some arid areas of western Rajasthan. Indian J. Med. Res. 103: 91-93.

Singh, K.V. and S.K. Bansal. 1996. Current status of *Anopheles stephensi* response to various insecticides in some areas of the Thar Desert. Indian J. Med. Res. 103: 299- 303.

Singh, K.V. and S.K. Bansal. 1996. Present susceptibility of *Culex tritaeniorhynchus* to conventional and some other insecticides in Kota (Rajasthan). J. Commun. Dis. 28: 64-66.

Srivastava, S.C. 1988. Susceptibility tests with certain insecticides against stable flies *Stomoxys calcitrans* L. (Diptera: Muscidae). Indian Vet. J. 65: 838-839.

Srivastava, P.K., D.N. Sarma, L. Dhanaraj and R.M. Sundaram. 1995. Larval susceptibility status of *Anopheles stephensi* against temephos in Madras, Tamil Nadu. J. Ecotoxicol. Environ. Monitor. 5: 139-142.

Subbarao, S.K., V.P. Sharma, K. Vasantha and T. Adak. 1984. Effect of malathion spraying on four anopheline species and the development of resistance in *A. stephensi* in Mandora, Haryana. Indian J. Malariol. 21: 109-114.

Subbarao, S.K., K. Vasantha and V.P. Sharma. 1988. Responses of *Anopheles culicifacies* sibling species A and B to DDT and HCH in India: implications in malaria control. Med. Vet. Entomol. 2: 219-223.

Sundaram, R.M., A.V. Sadan and G. Narayanasamy. 1989. Susceptibility status of vectors of Japanese encephalitis to insecticides in South Arcot and Tirunelveli Districts of Tamil Nadu. J. Commun. Dis. 21: 218-221.

Thapar, B.R., R.D. Joshi, J.S. Rao and N.B.L. Saxena. 1993. Susceptibility status of *Phlebotomus papatasi* Scopoli (Diptera: Culicidae) to chlorinated hydrocarbons in Panchamahal District of Gujarat State (India). J. Commun. Dis. 4: 212-213.

Thavaselvam, D., A. Kumar and P.K. Sumodan. 1993. Insecticide susceptibility status of *Anopheles stephensi*, *Culex quinquefasciatus* and *Aedes aegypti* in Panaji, Goa. Indian J. Malariol. 30: 75-79. (Published erratum appears in Indian J. Malariol. 30: 182.)

Thomas, G., S.K. Sharma, A. Prakash, J. Sokhey and B.R. Sharma. 2000. Insecticide susceptibility of *Culex tritaeniorhynchus* Giles, vector of Japanese encephalitis in Delhi. Jpn. J. Infect. Dis. 53: 11-14.

Vijayan, V.A. and N. Ningegowda. 1993. Susceptibility difference in two populations of *Culex quinquefasciatus* Say (Diptera: Culicidae) to three synthetic pyrethroids. S.E. Asian J. Trop. Med. Pub. Health 24: 540-543.

Vijayan, V.A. and N. Pushpalatha. 1997. A note on tolerance in *Culex vishnui*, a Japanese encephalitis vector. J. Commun. Dis. 29: 299-301.

Vijayan, V.A., M.A. Revanna, K.S. Vasudeva, N. Pushpalatha, and A. Poornima. 1993. Comparative susceptibility of two Japanese encephalitis vectors from Mysore to six insecticides. Indian J. Med. Res. 97: 215-217.

Vittal, M., R.B. Deobhankar and R.R. Deo. 1982. DDT resistance and seasonal variations in susceptibility to DDT and dieldrin in *Anopheles culicifacies* in Maharashtra. Pesticides 16: 25-26.

Vittal, M. and L.B. Deshpande. 1983. Development of malathion resistance in a DDT, HCH resistant *Anopheles culicifacies* population in Thane District (Maharashtra) J. Commun. Dis. 15: 144-145.

Vittal, M., S.M. Mustafa, R.B. Deobhankar, L.B. Deshpande and R.R. Deo. 1982. Insecticide susceptibility status of malaria vectors in Maharashtra. Indian J. Malariol. 19: 59-61.

Yadava, R.L., S.R. Dwivedi and R.G. Roy. 1989. Susceptibility status of *Culex vishnui* to DDT and dieldrin in Deoria, Uttar Pradesh. J. Commun. Dis. 21: 137-138.

Yadava, R.L., C.K. Rao and H. Biswas. 1996. Field trial of cyfluthrin as an effective and safe insecticide for control of malaria vectors in triple insecticide resistant areas. J. Commun. Dis. 28: 287-298.

Pakistan

Azmi, M.A., S.N.H. Naqvi and K. Akhtar. 1993. Comparative study of toxicity of DDT, malathion and Solfac against various strains of *Culex fatigans* by WHO method. Proc. Pakistan Congr. Zool. 13: 359-266.

Hemingway, J. 1982. The biochemical nature of malathion resistance in *Anopheles stephensi* from Pakistan. Pestic. Biochem. Physiol. 17: 149-155.

Hemingway, J. 1983. The genetics of malathion resistance in *Anopheles stephensi* from Pakistan. Trans. R. Soc. Trop. Med. Hyg. 77: 106-108.

Hussain, A., H.R. Rathor and Z. Hussain. 1995. DDT cross-resistance to permethrin in *Anopheles culicifacies* and *Anopheles stephensi* in Pakistan. Pakistan Vet. J. 15: 91-95.

Lewis, A.N. 1990. An assessment of how resistance to malathion by *Anopheles stephensi* affects the incidence of malaria in Pakistan. Vector Biol. Control Proj., Arlington, VA, AR-115-4, 42pp.

Mohiuddin, S. and Z. Ahmed. 1991. Comparative observation on the toxicity of some commonly used pesticides against laboratory reared and wild strains of *Aedes aegypti* (L). Pak. J. Sci. 34: 356-358.

Rathor, H.R. and G. Toqir. 1980. Malathion resistance in *Anopheles stephensi* Liston in Lahore, Pakistan. Mosq. News 40: 526-531.

Rathor, H.R. and G. Toqir. 1980. Selection for DDT susceptibility in *Anopheles culicifacies* from Lahore, Pakistan. Southeast Asian J. Trop Med. Pub. Health 11: 113-118.

Rathor, H.R., G. Toqir and S. Rashid. 1985. Insecticide resistance in anopheline mosquitoes of Punjab Province, Pakistan. Pak. J. Zool. 17: 35-49.

Rathor, H.R., G. Toqir, W.K. Reisen, S.M. Mujtaba and S.M. Nasir. 1980. Status of insecticide resistance in anopheline mosquitoes of Punjab Province, Pakistan. Southeast Asian J. Trop. Med. Pub. Health 11: 332-340.

Rawlings, P., G. Davidson, R.K. Sakai, H.R. Rathor, M. Aslamkhan and C.F. Curtis. 1981. Field measurement of the dominance of an insecticide resistance in anopheline mosquitos. WHO Bull. 59: 631-640.

Reisen, W.K. 1986. Population dynamics of some Pakistan mosquitoes: the impact of residual organophosphate insecticide spray on anopheline relative abundance. Ann. Trop. Med. Parasitol. 80: 69-75.

Rowland, M. 1985. Location of the gene for malathion resistance in *Anopheles stephensi* (Diptera: Culicidae) from Pakistan. J. Med. Entomol. 26: 373-380.

Rowland, M. and J. Hemingway. 1987. Changes in malathion resistance with age in *Anopheles stephensi* from Pakistan. Pestic. Biochem. Physiol. 28: 239-247.

Scott, J.G. and G.P. Georghiou. 1986. Malathion-specific resistance in *Anopheles stephensi* from Pakistan. J. Am. Mosq. Control Assoc. 2: 29-32.

Sri Lanka

Haskell, P.T., P.R.J. Herath and P.T. Haskell (ed.) 1982. Pesticide resistance and malaria control in Sri Lanka. *In*: Resistance to insecticides used in public health and agriculture. Proceedings of an international workshop held at the Sri Lanka Foundation Institute, Colombo, from 22 to 26 February 1982.

Hemingway, J. 1989. A note on simple biochemical methods for resistance detection and their field application in Sri Lanka. Pestic. Sci. 27: 281-285.

Hemingway, J., K.G. Jayawardena and P.R. Herath. 1986. Pesticide resistance mechanisms produced by field selection pressures on *Anopheles nigerrimus* and *A. culicifacies* in Sri Lanka. Bull. WHO 64: 753-758.

Hemingway, J., K.G. Jayawardena and I.S. Weerasinghe. 1988. The use of biochemical assays in the detection and characterization of insecticide resistance in field populations of mosquitoes. Excerpta Med. Int. Congr. Ser., Netherlands 810: 27.

Herath, P.R., J. Hemingway and I.S. Weerasinghe. 1987. The detection and characterization of malathion resistance in field populations of *Anopheles culicifacies* B in Sri Lanka. Pestic. Biochem. Physiol. 29: 157-162.

Herath, P.R. and G.P. Joshi. 1986. Factors affecting selection for multiple resistance in *Anopheles nigerrimus* in Sri Lanka. Trans. R. Soc. Trop. Med. Hyg. 80: 649-652.

Herath, P.R. and G.P. Joshi. 1989. Pesticide selection pressure on *Anopheles subpictus* in Sri Lanka: comparison with two other Sri Lankan anophelines. Trans. R. Soc. Trop. Med. Hyg. 83: 565-567.

Karunaratne, S.H. 1999. Insecticide cross-resistance spectra and underlying resistance mechanisms of Sri Lankan anopheline vectors of malaria. Southeast Asian J. Trop. Med. Pub. Health 30: 460-469.

Karunaratne, S.H., K.G. Jayawardena, J. Hemingway and A.J. Ketterman. 1993. The function of esterases in insecticide resistance in *Culex quinquefasciatus* mosquitoes from Sri Lanka. Biochem. Soc. Trans. 21: 482S

Karunaratne, S.H.P.P., A. Vaughan, M.G. Paton and J. Hemingway. 1998. Amplification of a serine esterase gene is involved in insecticide resistance in Sri Lankan *Culex tritaeniorhynchus*. Insect Mol. Biol. 7: 307-315.

Peiris, H.T.R. and J. Hemingway. 1990. Temephos resistance and the associated cross-resistance spectrum in a strain of *Culex quinquefasciatus* Say (Diptera: Culicidae) from Peliyagoda, Sri Lanka. Bull. Entomol. Res. 80: 49-55.

Peiris, H.T.R. and J. Hemingway. 1990. Mechanisms of insecticide resistance in a temephos selected *Culex quinquefasciatus* (Diptera: Culicidae) strain from Sri Lanka. Bull. Entomol. Res. 80: 453-457.

Peiris, H.T.R. and J. Hemingway. 1996. Effect of fenthion on larval densities of insecticide-resistant *Culex quinquefasciatus* in an urban area of Sri Lanka. Med. Vet. Entomol. 10: 283-287.

Pinikahana, J. and R.A. Dixon. 1993. Trends in malaria morbidity and mortality in Sri Lanka. Indian J. Malariol. 30: 51-55.

Rawlings, P., P.R. Herath and S. Kelly. 1985. *Anopheles culicifacies* (Diptera: Culicidae) DDT resistance in Sri Lanka prior to and after cessation of DDT spraying. J. Med. Entomol. 22: 361-365.

* Only papers published in the 20 years prior to preparation of this document are included. Many of these articles describe tests on insect populations that were found to be susceptible or contain general discussions about pesticide resistance in South Central Asia.

Appendix D. Sources of Snake Antivenoms

1	Perusahaam Negara Biofarms 9, Jalan Pasteur Bandung, Indonesia
2	Behring Institut, Behringwerke AG, D3550 Marburg (Lahn), Postfach 167, Germany. Telephone: (06421) 39-0. Telefax: (06421) 660064. Telex: 482320-02
3	Institute of Epidemiology and Microbiology, Sofia, Bulgaria
4	Shanghai Vaccine and Serum Institute, 1262 Yang An Road (W), Shanghai, PRC
5	Commonwealth Serum Laboratories, 45 Poplar Road, Parkville, Victoria 3052, Australia Telegram: "SERUMS," Melbourne Telex: AA32789, Telephone: 387-1066
6	Foreign Trade Company, Ltd., Kodandaka, 46 Prague 10, Czech Republic
7	Fitzsimmons Snake Park, Box 1, Snell Park, Durban, South Africa
8	Haffkine Bio-pharmaceutical Corporation, Ltd., Parel, Bombay, India
9	Chiba Serum Institute, 2-6-1 Konodai, Ichikawa, Chiba Prefecture, Japan
10	Institut d'État des Serums et Vaccins Razi, P.O. Box 656, Tehran, Iran
11	Central Research Institute, Kasauli (Simia Hills), (H.P.) India
12	Kitasato Institute, 5-9-1 Shirokane, Minato-ku, Tokyo, Japan
13	The Chemo-Sero Therapeutic Research Institute, Kumamoto, 860 Kyushu, Japan
14	National Institute of Health, Biological Production Division, Islamabad, Pakistan. Telex: 5811-NAIB-PK, Telephone: 820797, 827761
15	Research Institute For Microbial Diseases, Osaka University, 3-1 Yamadoaka, Suite 565, Osaka, Japan, Telephone: (06) 877-5121
16	Institut Pasteur Production, 3 Boulevard Raymond Poincaré, 92430-Mames la Coquette, France. Telephone: (1) 47.41.79.22, Telex: PASTVAC206464F
17	Institut Pasteur d'Algérie Docteur Laveran, Algiers, Algeria
18	Industrial and Pharmaceutical Corporation, Rangoon, Burma
19	Rogoff Medical Research Institute, Beillinson Medical Center, Tel-Aviv, Israel
20	South African Institute for Medical Research, P.O. Box 1036, Johannesburg 2000, Republic of South Africa. Telegraph: "BACTERIA", Telephone: 724-1781
21	Instituto Sieroterapica e Vaccinogeno Toscano "Sclavo", Via Fiorentina 1, 53100 Siena, Italy.
22	National Institute of Preventive Medicine, 161 Kun-Yang St., Nan-Kang, Taipei, Taiwan
23	Takeda Chemical Industries, Ltd., Osaka, Japan
24	Research Institute of Vaccine and Serum, Ministry of Public Health U.I. Kafanova, 93 Tashkent, USSR
25	Red Cross Society, Queen Saovabha Memorial Institute, Rama 4 Road, Bangkok, Thailand 26
26	Twyford Pharmaceutical Services Deutschland, GmbH, Postfach 2108 05, D-6700 Ludwigshafen am Rhein, Germany
27	Institute of Immunology, Rockefellerova 2, Zagreb, Yugoslavia

Appendix E. Selected List of Taxonomic Papers and Identification Keys*

Centipedes and Millipedes

Jangi, B.S. 1966. *Scolopendra*. (The Indian centipede). *In*: Indian zoological memoirs, No. 9. Zool. Soc. India, Calcutta: 109 pp.*****

Ceratopogonidae

Atchley, W.R., W.W. Wirth and C.T. Gaskins. 1981. A bibliography and keyword index of the biting midges (Diptera: Ceratopogonidae). USDA Sci. Educ. Admin. Bibliogr. Lit. Agric. No. 13: 1-544.

Giles, F.E., W.W. Wirth and D.H. Messersmith. 1981. Two new species of biting midges and a checklist of the genus *Culicoides* (Diptera: Ceratopogonidae) from Sri Lanka. Proc. Entomol. Soc. Wash. 83: 537-543.

Kitaoka, S. and S. Shinonaga. 1989. Biting midges from northern Pakistan with descriptions of three new species (Diptera: Ceratopogonidae). Jap. J. Sanit. Zool. 40: 25-31.

Szadziewski, R. and P. Havelka. 1984. A review of the Palearctic biting midges of the subgenus *Brachypogon* (s. str.) (Diptera, Ceratopogonidae). Pol. Pismo Entomol. 54: 341-358.*****

Wirth, W.W. and A.A. Hubert. 1989. The *Culicoides* of Southeast Asia (Diptera: Ceratopogonidae). Mem. Am. Entomol. Inst. No.44: 1-511.*****

Chiggers

Nadchatram, M. 1970. Nepal chiggers I. Species of the genus and subgenus *Leptotrombidium*, with synonymic notes (Prostigmata: Trombiculidae). J. Med. Entomol. 7: 145-163.

Cimicidae

Bhat, H.R. 1974. A review of Indian Cimicidae (Hemiptera-Heteroptera). Orient. Insects 8: 545-550.

Ryckman, R.E., D.G. Bentley and E.F. Archbold. 1981. The Cimicidae of the Americas and Oceanic Islands, a checklist and bibliography. Bull. Soc. Vector Ecol. 6: 93-142.

Culicidae

Abhayawardana, T.A., S. R. Wijesuriya and R.K.C. Dilrukshi. 1996. *Anopheles subpictus* complex: distribution of sibling species in Sri Lanka. Indian J. Malariol. 33: 53-60.

Ahmed, T.U. 1987. Checklist of the mosquitoes of Bangladesh. Mosq. Syst. 19: 187-200.

Amerasinghe, F.P. 1992. A guide to the identification of the anopheline mosquitoes (Diptera: Culicidae) of Sri Lanka. II. Larvae. Cey. J. Sci. 22: 1-13.*****

Amerasinghe, F.P. 1996. Keys for the identification of the immature stages of genus *Culex* (Diptera: Culicidae) in Sri Lanka. J. Natl. Sci. Coun. Sri Lanka 24: 37-50.*****

Amerasinghe, F.P., N.B. Munasingha and V.L. Kulasekera. 1987. Some new records of mosquitoes occurring in Sri Lanka. Mosq. Syst. 19: 162-166.

Danilov, V.N. 1985. Mosquitoes (Diptera, Culicidae) of Afghanistan. Contribution 1. Key to females. Med. Parazitol. 2: 67-72.*****

Danilov, V.N. 1985. Mosquitoes (Diptera, Culicidae) of Afghanistan. Contribution 2. Key to 4th stage larvae. Med. Parazitol. 4: 51-55.*

Darsie, R.F. Jr. and S.P. Pradhan. 1987. A visit to USAID/Nepal to study and identify culicine fauna. Vector Biol. Control Proj., Arlington, VA AR-061, 41 pp.*

Darsie, R.F. Jr. and S.P. Pradhan. 1990. The mosquitoes of Nepal: their identification, distribution and biology. Mosq. Syst. 22: 69-130.

Darsie, R.F. Jr. and S.P. Pradhan. 1991. The mosquitoes of Nepal, their identification, distribution and biology. Index and corrigendum. Mosq. Syst. 23: 46-49.

Darsie, R.F. Jr., S.P. Pradhan and R.G. Vaidya. 1991. Notes on the mosquitoes of Nepal. I. New country records and revised *Aedes* keys (Diptera: Culicidae). Mosq. Syst. 23: 39-45.*

Das, B.P. and S.M. Kaul. 1998. Pictorial key to the common Indian species of *Aedes* (*Stegomyia*) mosquitoes. J. Commun. Dis. 30: 123-127.*

Dobrotovsky, N. 1971. The genus *Culiseta* Felt in Southeast Asia. Contrib. Am. Entomol. Inst. 7: 38-61.*

Gaffigan, T.V. and R.A. Ward. 1985. Index to the second supplement to “A catalog of the mosquitoes of the world (Diptera: Culicidae).” Mosq. Syst. 17: 52-63.

Glick, J.I. 1992. Illustrated key to the female *Anopheles* of southwestern Asia and Egypt (Diptera: Culicidae). Mosq. Syst. 24: 125-153.*

Harbach, R.E. 1985. Pictorial keys to the genera of mosquitoes, subgenera of *Culex* and the species of *Culex* (*Culex*) occurring in southwestern Asia and Egypt, with a note on the subgeneric placement of *Culex deserticola* (Diptera: Culicidae). Mosq. Syst. 17: 83-107.*

Harrison, B. and J. Reinert. 1974. Distributional and biological notes on mosquitoes from Sri Lanka (Diptera: Culicidae). Mosq. Syst. 6: 142-162.

Huang, Y.M. 1979. Medical entomology studies. XI. The subgenus *Stegomyia* of *Aedes* in the Oriental region with keys to the species (Diptera: Culicidae). Contrib. Am. Entomol. Inst. 15: 1-79.*

Knight, K.L. 1978. Supplement to “A catalog of the mosquitoes of the world (Diptera: Culicidae).” Thomas Say Foundation, Entomological Society of America, Vol. 6, 107 pp.

Knight, K.L. and A. Stone. 1977. A catalog of the mosquitoes of the world (Diptera: Culicidae). 2nd ed. Thomas Say Foundation, Entomological Society of America, Vol. 6, 611 pp.

Malhotra, P.R. and H.C. Mahanta. 1994. Checklist of mosquitoes of northeast India (Diptera: Culicidae). Orient. Insects 28: 125-149.

Mattingly, P. 1971. Contributions to the mosquito fauna of Southeast Asia. XIII. Illustrated keys to the genera of mosquitoes (Diptera: Culicidae). Contrib. Am. Entomol. Inst. 7: 1-84.*

Pradhan, S.P. and R.F. Darsie Jr. 1989. New mosquitoes records for Nepal. J. Am. Mosq. Control Assoc. 5: 21-24.

Rajavel, A.R. 1996. Taxonomic notes on *Culex* (*Lophoceraomyia*) *infantulus* (Diptera: Culicidae) based on its new distribution record in Pondicherry, South India. Entomon 21: 43-48.*

Rao, T.R. 1984. The anophelines of India. Indian Council of Medical Research, New Delhi. Revised Edition. 518 pp.*****

Reinert, J.F. 1984. Medical entomology studies - XVI. A review of the species of subgenus *Verrallina*, genus *Aedes*, from Sri Lanka and a revised description of the subgenus (Diptera: Culicidae). Mosq. Syst. 16: 1-2.*****

Reuben, R., S.C. Tewari and J. Hiriyan. 1994. Illustrated keys to species of *Culex* (*Culex*) associated with Japanese encephalitis in Southeast Asia (Diptera: Culicidae). Mosq. Syst. 26: 75-96.*****

Srivastava, A., R. Saxena, B. Nagpal and V.P. Sharma.. 1992. Matrix based approach for identification of Indian anophelines. Indian J. Malariol. 29: 185-191.

Tewari, C., J. Hiriyan and R. Reuben. 1987. Survey of the anopheline fauna of the Western Ghats in Tamil Nadu, India. Indian J. Malariol. 24: 21-28.

Tyson, W. 1970. Contributions to the mosquito fauna of Southeast Asia. VIII. Genus *Aedes*, subgenus *Mucidu* Theobald in Southeast Asia. Contrib. Am. Entomol. Inst. 6: 29-80.*****

Ward, R.A. 1984. Second supplement to "A catalog of the mosquitoes of the world (Diptera: Culicidae)." Mosq. Syst. 16: 227-270.

Ward, R.A. 1992. Third Supplement to "A catalog of the mosquitoes of the world (Diptera: Culicidae)." Mosq. Syst. 24: 177-230.

Zavortink, T. 1971. The genus *Orthopodomyia* in Southeast Asia. Contrib. Am. Entomol. Inst. 7: 1-37.

Other Diptera

Nishida, K. 1989. The Fanniidae from Pakistan (Diptera). Jap. J. Sanit. Zool. 40: 87-89.*****

Kurahashi, H. and Q. Banu. 1989. Notes on the Bangladesh calliphorid flies of medical importance (Insecta: Diptera). Jap. J. Sanit. Zool. 40: 97-111.*****

Pont, A.C. 1977. Family Muscidae. pp 451-523 *In*: A catalog of the Diptera of the Oriental region. Volume III. Suborder Cyclorrhapha (excluding division Aschiza). M.D. Delfinado (ed.). University Press of Hawaii, Honolulu.

Shinonaga, S., H. Kurahashi and M. Iwasa. 1994. Studies on the taxonomy, ecology and control of the medically important flies in India and Nepal. Jap. J. Sanit. Zool. 45(suppl.) 316pp.*****

Sugiyama, E. 1989. Sarcophagine flies from Pakistan (Diptera, Sarcophagidae). Jap. J. Sanit. Zool. 40: 13-124.*****

Sugiyama, E., S. Shinonaga and R. Kano. 1988. Sarcophagine flies from Nepal with the description of a new species (Diptera: Sarcophagidae). Jap. J. Sanit. Zool. 39: 355-362.*****

Mammalia

Nowak, R.M. (ed.) 1999. Walker's Mammals of the World. 6th ed., John Hopkins University Press, Baltimore, 1629 pp.

Wilson, D.E. and D.M. Reeder. 1993. Mammal Species of the World: a Taxonomic and Geographic Reference. 2nd ed., Smithsonian Institution Press, Washington, DC, 1,206 pp.

Psychodidae

Artemiev, M.M. 1974. Sandflies (Diptera, Psychodidae, Phlebotomidae) of Afghanistan. Communication I. The genus *Phlebotomus*. Med. Parazitol. 43: 154-165.

Artemiev, M.M. 1974. Sandflies (Diptera, Psychodidae, Phlebotomidae) of eastern Afghanistan. Communication II. The genus *Sergentomyia*. Med. Parazitol. 43: 328-334.

Artemiev, M.M. 1976. Sandflies (Diptera, Psychodidae, Phlebotomidae) of east Afghanistan. Communication III. The genus *Sergentomyia* , subgenera *Sintonius*, *Rondanomyia* and *Grassomyia*. Med. Parasitol. 45: 35-41.

Artemiev, M.M. 1976. Sandflies (Diptera, Psychodidae, Phlebotomidae) of eastern Afghanistan. Communication IV. The genus *Sergentomyia*, the subgenus *Parrotomyia*. Med. Parasitol. 45: 422-429.

Hossain, M.I., S.A. Kahn and M. Ameen. 1993. Phlebotomine sandflies of Bangladesh: recent surveys. Med. Vet. Entomol. 7: 99-100.

Ilango, K., V.D.R. Spinivasan, A.V. Sadanand and R.P. Lane. 1994. Phlebotomine sandflies (Diptera: Psychodidae) of Tamil Nadu and Pondicherry, southern India, in relation to visceral leishmaniasis. Ann. Trop. Med. Parasitol. 88: 413-431.

Javadian, E., A. Nadim and A.K. Nayil. 1982. Epidemiology of cutaneous leishmaniasis in Afghanistan. Part III. Notes on sandflies of Afghanistan. Bull. Soc. Pathol. Exot. 75: 284-290.

Kaul, S.M. 1993. Phlebotomine sandflies (Diptera: Psychodidae) from Western Ghats in Kerala and Tamil Nadu States, India. Part II: taxonomic and biological notes on the species recorded. J. Commun. Dis. 25: 116-125.

Lewis, D.J. 1967. The phlebotomine sandflies of West Pakistan (Diptera: Psychodidae). Bull. Brit. Mus. Nat. History, Entomology Series, 19: 1-57.*****

Lewis, D.J. 1973. Family Phlebotomidae. pp 245-254 *In*: A catalog of the Diptera of the Oriental region. Volume I. Suborder Nematocera. M.D. Delfinado (ed.). Univ. Press Hawaii, Honolulu.

Lewis, D.J. 1978. The phlebotomine sandflies (Diptera : Psychodidae) of the Oriental region. Bull. Brit. Mus. Nat. History, Entomology Series 37: 1-343.*****

Lewis, D.J. 1982. A taxonomic review of the genus *Phlebotomus* (Diptera: Psychodidae). Bull. Brit. Mus. Nat. History, Entomology Series 45: 1- 209.*

Lewis, D.J. 1987. Phlebotomine sandflies (Diptera: Psychodidae) from the Oriental region. Syst. Entomol. 12: 163-180.*****

Seyedi-Rashti, M.A., A. Nadim, M.A. Rashti and M.A. Sayedi. 1992. The genus *Phlebotomus* (Diptera: Psychodidae : Phlebotominae) of the countries of the eastern Mediterranean region. Iranian J. Pub. Hlth. 21: 11-50.*****

Reptiles

Murthy, T.S.N. 1985. Classification and distribution of the reptiles of India. Snake 17: 48-70.

Harding, K.A. and K.R.G. Welch. 1980. Venomous Snakes of the World: a Checklist. Pergamon Press, Oxford, 188 pp.

McDiarmid, R., J. Campbell and T. Toure. 1999. Snake species of the world. A taxonomic and geographic reference. Volume I. The Herpetologist’s League, Washington, DC, 511 pp.

Poisonous Snakes of the World, a Manual for Use by U.S. Amphibious Forces. 1966. NAVMED P-5099, BUMED, Department of the Navy, U.S. Gov. Print. Off., 212 pp.*

Ratnapala, R. 1983. Key to the medically important venomous snakes of Sri Lanka. Ceylon Med. J. 28: 129-130.*

Welch, K.R.G. 1988. Snakes of the Orient: a checklist. Robert E. Krieger Publishing Co., Malabar, FL, 188pp.

Scorpions

El Hennawy, H.K. and H.K. El Hennawy. 1990. Key to scorpion families (Arachnida: Scorpionida). Serket 2: 14-19.*

Keegan, H.L. 1980. Scorpions of medical importance. Univ. Press Miss., Jackson, 140 pp.

Sissom, W.D. 1990. Systematics, Biogeography, and Paleontology. pp 64-136 *In:* The Biology of Scorpions, G.A. Polis (ed.). Stanford University Press.*

Stahnke, H.L. 1972. A key to the genera of Buthidae (Scopionida). Ent. News 83: 121-133.*

Simuliidae

Datta, M. 1974. Some black flies (Diptera: Simuliidae) of the subgenus *Simulium* Latreille (s. str.) from the Darjeeling area. Orient. Insects 8: 15-27.

Datta, M. 1992. An overview of the Simuliidae (Diptera) of West Bengal, India. J. Bengal Nat. Hist. Soc. 11: 41-62.

Davies, D.M. and H. Gyorkos. 1987. The Simuliidae (Diptera) of Sri Lanka. Description of species in the subgenera *Eusimulium* and *Gomphostilbia* of the genus *Simulium*. Can. J. Zool. 65: 1483-1502.*

Davies, D.M. and H. Gyorkos. 1992. The Simuliidae (Diptera) of Sri Lanka. Descriptions of additional species of *Simuliun* (*Simulium*), with a key for Sri Lankan species in the subgenus and a checklist for the country. Can. J. Zool. 70: 1029-1046.*

Kim, K.C. and R.W. Merritt. 1987. Black flies - ecology, population management, and annotated world list. PA State Univ., University Park, 543 pp.

Lewis, D.J. 1973. The Simuliidae (Diptera) of Pakistan. Bull. Entomol. Res. 62: 453-470.*

Saito, K., K. Uemoto and M. Afzal. 1989. The black flies (Diptera: Simuliidae) collected in the northern part of Pakistan. Jap. J. Zool. 40: 33-40.

Takaoka, H. 1996. The geographical distribution of the genus *Simulium* Latreille in the Oriental and Australian regions. Jap. J. Trop. Med. Hyg. 24: 113-124.

Siphonaptera

Adams, N.E. and R.E. Lewis. 1995. An annotated catalogue of primary types of Siphonaptera in the National Museum of Natural History, Smithsonian Institution. Smithsonian Contributions to Zoology. Number 56: 1-86.

Arsenjeva, L. and V. Neronov. 1982. Materials on the fauna of fleas from Afghanistan. Parasitologiya 16: 306-314.

Iyengar, R. 1973. The Siphonaptera of the Indian subregion. Oriental Insects. Supplement N0. 3. 102 pp.

Lewis, R.E. 1971. New neopsylline fleas from Nepal (Neopsyllinae: Hystrichopsyllidae). J. Parasitol. 57: 401-416.

Lewis, R.E. 1973. Siphonaptera collected during the 1965 Street expedition to Afghanistan. Fieldiana Zool. 64: 1-161.

Lewis, R.E. 1981. A review of the genus *Amphipsylla* in Nepal, Pakistan and northern India, with the description of a new taxon and a key to the species treated (Siphonaptera: Leptopsyllidae). J. Med. Entomol. 18: 48-60.*****

Lewis, R.E. 1990. The Ceratophyllidae: currently accepted valid taxa (Insecta: Siphonaptera). Theses Zoologicae, volume 13. R. Fricke (ed.). Koeltz Scientific Books. Koenigstein, Germany. 267 pp.

Lewis, R.E. and J.H. Lewis. 1985. Notes on the geographical distribution and host preferences in the order Siphonaptera. Part 7. New taxa described between 1972 and 1980, with a supraspecific classification of the order. J. Med. Entomol. 22: 134-152.

Lewis, R.E. and J.H. Lewis. 1989. Catalogue of invalid genus-group and species-group names in the Siphonaptera (Insecta). These Zoologicae, Volume 11. R. Fricke (ed.). Koeltz Scientific Books. Koenigstein, Germany. 263 pp.

Lewis, R.E. 1998. Résumé of the Siphonaptera (Insecta) of the world. J. Med. Entomol. 35: 377-389.

Smit, F.G.A.M. and B. Rosicky. 1974. Siphonaptera from eastern Afghanistan. Folia Parasitol. 21: 29-37.

Traub, R. 1969. Table 6. Tentative identifications of Siphonaptera collected in Nepal by field teams of the Bishop Museum. pp. 138-155. *In*: Nepal Health Survey 1965-1966. R.M. Worth and N.K. Shah (eds). Univ. Press Hawaii, Honolulu.

Traub, R., M. Rothschild and J.F. Haddow. 1983. The Rothschild Collection of Fleas. The Ceratophyllidae: Key to the Genera and Host Relationships. With notes on Their Evolution, Zoogeography and Medical Importance. Privately published by M. Rothschild and R. Traub. Distributed by Academic Press. 288 pp.

Spiders

Tikader, B.K. 1976. Key to Indian spiders. J. Bombay Nay. Hist. Soc. 73: 356-370.*****

Tabanidae

Coher, E.I. 1985. Asian biting fly studies IV: Tabanidae (Diptera). Records and new species from Nepal with additional notes on some forms from Sikkim and Assam. Myia 3: 251-275.

Stone, A. and C.B. Philip. 1974. The Oriental species of the tribe Haematopotini (Diptera, Tabanidae). USDA ARS Tech. Bull. No. 1489: 240pp.*****

Ticks (Ixodidae, Argasidae)

Balashov, Yu.S. 1972. Bloodsucking ticks (Ixodoidea) - vectors of diseases of man and animals. Bull. Entomol. Soc. Amer. 8: 161 - 376.

Clifford, C.M., H. Hoogstraal and J.E. Keirans. 1975. The *Ixodes* ticks (Acarina: Ixodidae). of Nepal. J. Med. Entomol. 12: 115-137.

Geevarghese, G. and V. Dhanda. 1987. The Indian *Hyalomma* ticks (Ixodoidea: Ixodidae). Indian Counc. Agric. Res., New Delhi. 119 pp.*****

Kaiser, M. and H. Hoogstraal. 1963. The *Hyalomma* ticks (Ixodoidae: Ixodidae) of Afghanistan. J. Parasitol. 49: 130-139.

Kaiser, M. and H. Hoogstraal. 1964. The *Hyalomma* ticks (Ixodoidae: Ixodidae) of Pakistan, India and Ceylon with keys to subgenera and species. Acarologia 6: 257-286.*****

Miranpuri, G.S. and H.S. Gill. 1983. Ticks of India. Forward by D.W. Brocklesby. Centre for Tropical Veterinary Medicine, University of Edinburgh, Edinburgh, Scotland. pp.147.

Seneviratna, P. 1965. The Ixodoidea (ticks) of Ceylon. Parts II and III. Ceylon Vet. J. 13: 28-54*****

Sharif, M. 1928. A revision of the Indian Ixodidae with special reference to the collection in the Indian Museum. Records of the Indian Museum 30: 217-344.*****

Siddiqi, M.N. and A.H. Jan. 1986. Ixodid ticks (Ixodidae) of N.W.F.P. (Pakistan). Pak. Vet. J. 6: 124-126.

Trapido, H., M.G.R. Varma, P.K. Rajagopalan, K.R.P. Singh and M.J. Rebello. 1964. A guide to the identification of all stages of the *Haemaphysalis* ticks of south India. Bull. Ent. Res. 55: 249-270.

Walker, J.B., J.E. Keirians and I.G. Horak. 2000. The genus *Rhipicephalus* (Acari, Ixodidae): A guide to the brown ticks of the world. Cambridge University Press.

*Papers marked with an asterisk include a taxonomic key for identification of species. References without keys for identification usually contain a checklist of species known from a given geographic area or a list of species collected during extensive surveys of an area.

Appendix F. Personal Protective Measures

Personal protective measures are the first line of defense against arthropod-borne disease and, in some cases, may be the only protection for deployed military personnel. Proper wearing of the uniform and appropriate use of repellents can provide high levels of protection against blood-sucking arthropods. The uniform fabric provides a significant mechanical barrier to mosquitoes and other blood-sucking insects. Therefore, the uniform should be worn to cover as much skin as possible if weather and physical activity permit. When personnel are operating in tick-infested areas, they should tuck their pant legs into their boots to prevent access to the skin by ticks, chiggers, and other crawling arthropods. They should also check themselves frequently for ticks and immediately remove any that are found. If a tick has attached, seek assistance from medical authorities for proper removal or follow these guidelines from TIM 36, Section IX A.

1. **Grasp the tick's mouthparts** where they enter the skin, using pointed tweezers.

2. **Pull out** slowly and steadily with gentle force.

    a. Pull in the reverse of the direction in which the mouthparts are inserted, as you would for a splinter.

    b. **Be patient** – The long, central mouthpart (called the hypostome) is inserted in the skin. It is covered with sharp barbs, sometimes making removal difficult and time consuming.

    c. Many hard ticks secrete a cement-like substance during feeding. This material helps secure their mouthparts firmly in the flesh and adds to the difficulty of removal.

    d. It is important to continue to pull steadily until the tick can be eased out of the skin.

    e. **Do not** pull back sharply, as this may tear the mouthparts from the body of the tick, leaving them embedded in the skin. If this happens, don't panic. Embedded mouthparts are comparable to having a splinter in your skin. However, to prevent secondary infection, it is best to remove them. Seek medical assistance if necessary.

    f. **Do not** squeeze or crush the body of the tick because this may force infective body fluids through the mouthparts and into the wound.

    g. **Do not** apply substances like petroleum jelly, fingernail polish remover, repellent pesticides, or a lighted match to the tick while it is attached. These materials are either ineffective or, worse, may agitate the tick and cause it to salivate or regurgitate infective fluid into the wound site.

    h. If tweezers are not available, grasp the tick's mouthparts between your fingernails, and remove the tick carefully by hand. Be sure to wash your hands -- especially under your fingernails -- to prevent possible contamination by infective material from the tick.

3. Following removal of the tick, **wash the wound** (and your hands) with soap and water and **apply an antiseptic**.

4. **Save the tick** in a jar, vial, small plastic bag, or other container for identification, should you later develop disease symptoms. Preserve the tick by either adding some alcohol to the jar or by keeping it in a freezer. Storing a tick in water will not preserve it. Identification of the tick will help the physician's diagnosis and treatment, since many tick-borne diseases are transmitted only by certain species.

5. **Discard** the tick after one month; all known tick-borne diseases will generally display symptoms within this time period.

Newly developed repellents provide military personnel with unprecedented levels of protection. An aerosol formulation of permethrin (NSN 6840-01-278-1336) can be applied to the uniform according to label directions, but not to the skin. This will impart both repellent and insecticidal properties to the uniform material that will be retained through numerous washings. An extended formulation lotion of N, N-diethyl-m-toluamide (deet) (NSN 6840-01-284-3982) has been developed to replace the 2 oz. bottles of 75% deet in alcohol. This lotion contains 33% active ingredient. It is less irritating to the skin, has less odor and is generally more acceptable to the user. A properly worn Battle Dress Uniform (BDU) impregnated with permethrin, combined with use of extended duration deet on exposed skin, has been demonstrated to provide nearly 100% protection against a variety of blood-sucking arthropods. This dual strategy is termed the DoD ARTHROPOD REPELLENT SYSTEM. In addition, permethrin may be applied to bednets, tents, and other field items as appropriate. Complete details regarding these and other personal protective measures are provided in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance (2000).

Appendix G. Bioscience and State Department Contacts in South Central Asia.

1. Regional Contacts.

World Health Organization (WHO) Headquarters Office in Geneva (HQ) Avenue Appia 20 1211 Geneva 27, SWITZERLAND	Phone: +00-41-22-791-2111 FAX: +00-41-22-791-3111 Website: < http://www.who.int/home/hq.html >
World Health Organization (WHO) Regional Office for the Eastern Mediterranean (EMRO) P.O. Box 1517 Alexandria – 21511 EGYPT [for: Afghanistan and Pakistan]	Phone: 203-48-202-23 *or* 203-48-202-24 FAX: 203-48-389-16 e-mail: < emro@who.sci.eg > Website: < http://www.who.sci.eg >
World Health Organization (WHO) Regional Office for South-East Asia (SEARO) World Health House Indraprastha Estate Mahatma Gandhi Road New Delhi 110002, INDIA [for: Bangladesh, Bhutan, India, Maldives, Nepal, and Sri Lanka]	Phone: 91-11-331-7804 *or* 91-11-331-7823 FAX: 91-11-331-8607 *or* 91-11-331-7972 e-mail: < postmaster@whosea.org > Website: < http://www.whosea.org >
Centers for Disease Control and Prevention Division of Quarantine National Center for Infectious Diseases 1600 Clifton Road, NE Atlanta, GA 30333 U.S.A. Travelers' Health Hotline: 877-FYI-TRIP (= 394-8747); FAX: 888-232-3299	Phone: (404) 639-3311 e-mail: < netinfo@cdc.gov > Website: <http://www.cdc.gov/travel/index.htm >
Commander U.S. Army Medical Component AFRIMS APO, AP 96546	Phone: 66-2-644-5777 (Thailand) FAX: 66-2-247-6030 (Thailand)
Commanding Officer NAMRU-2 Box 3, Unit 8132 APO, AP 96520-8132	Phone: 011-62-21-421-4457 (Jakarta, INDONESIA) or 011-62-21-421-4463 FAX: 011-62-21-421-4407 (Jakarta, INDONESIA)
Commanding Officer NAMRU-3 PSC 452, Box 131 FPO, AE 09835-0007	Phone: 011-20-2-284-1381 (Cairo, EGYPT) FAX: 011-20-2-284-1381 (Cairo, EGYPT)

2. Afghanistan.

The U.S. Embassy in Kabul has been closed since January 1989 for security reasons.

The Pakistani embassy operations in the U.S. were suspended August 21, 1997.

Appendix G. Bioscience and State Department Contacts in South Central Asia.

1. Regional Contacts.

Chancery:	Phone: 202-234-3770
2341 Wyoming Avenue, NW	FAX: 202-3328-3516
Washington, DC 20008	
Consulate(s) General: New York	

3. Bangladesh.

Ambassador	Phone: 880-2-884-700 (through -722)
Diplomatic Enclave	FAX: 880-2-883-744
Madani Ave.	
Baridhara, Dhaka	
BANGLADESH	

mailing address: G.P.O. Box 323, Dhaka 1000, BANGLADESH

4. Bhutan.

The U.S. and Bhutan have no formal diplomatic relations, but informal contact is maintained between the Bhutanese and U.S. Embassies in New Delhi, INDIA.

5. India.

Ambassador	Phone: 91-11-688-9033
Shanti Path	*or* 91-11-611-3033
Chanakyapuri 110021	FAX: 91-11-419-0017
New Delhi, INDIA	
U.S. Consulate General - Calcutta	FAX: 33-242-2335
5/1, Ho Chi Minh Sarani	Phone: 33-242-3611, 33-242-3615
Calcutta 700071	
U.S. Consulate General - Chennai	Phone: 44-827-7835, 44-827-3040
(Madras)	FAX: 44-825-0240
Mount Road	
Chennai 600006	
U.S. Consulate General - Mumbai	Phone: 22-363-3611, 22-363-3618
(Bombay)	FAX: 22-363-0350
Lincoln House	
78 Bhulabhai	
Desai Road	
Mumbai 400026	

6. Maldives.

The U.S. does not have an embassy in Maldives; the U.S. Ambassador to Sri Lanka is accredited to Maldives and makes periodic visits there.

Maldives does not have an embassy in the U.S. but does have a Permanent Mission to the U.N. in New York.

7. Nepal.

Ambassador	Phone: 977-1-411-179
Pani Pokhari	FAX: 977-1-419-963
Kathmandu, NEPAL	

U.S. Mission Home Page, Kathmandu, NEPAL: < http://www.south-asia.com/USA >

Appendix G. Bioscience and State Department Contacts in South Central Asia.

1. Regional Contacts.

8. Pakistan.

Ambassador Diplomatic Enclave, Ramna 5 Islamabad, PAKISTAN	Phone: 92-51-826-161 (through -179) FAX: 92-51-276-427
American Consulate - Karachi 8, Abdullah Haroon Road Karachi, PAKISTAN	Phone: 92-21-5685170 (through -5685179) FAX: 92-21-5683089
American Consulate - Lahore 50, Empress Road Lahore, PAKISTAN	Phone: 92-42-6365530 (through -6365539) FAX: 92-42-6365177
American Consulate - Peshawar 11, Hospital Road Peshawar, PAKISTAN	Phone: 92-91-279801 (through -279803) FAX: (2-91-276712

9. Sri Lanka.

Ambassador 210 Galle Road Colombo 3, SRI LANKA	Phone: 94-1-448-007 FAX: 94-1-437-345, *or* 94-1-446-013

mailing address: P.O. Box 106, Colombo, SRI LANKA

Appendix H: Glossary

acaricide - a substance developed to kill ticks and mites.
adulticide - insecticides used to kill the adult stages of an insect.
anaphylaxis - an unusual and severe allergic reaction of an organism to a foreign protein or other substances.
anthropophilic - the preference of insects and other arthropods to suck blood from humans rather than from animals.
autochthonous - transmission of a disease in the place where the disease occurred.
autogenous - not requiring a bloodmeal to produce eggs.
bionomics - the ecology of an organism.
biotope - a habitat characterized by environmental conditions and its community of animals and plants.
campestral - relating to fields or open country.
carrier - a person or animal that harbors infectious organisms but is free of clinical disease; generally synonymous with reservoir.
case fatality rate - the percentage of persons diagnosed as having a specific disease who die as a result of that illness within a given period.
cephalothorax - a body region consisting of head and thoracic segments.
cercaria - (pl. cercariae) - free-living stage in the life cycle of *Schistosoma* that emerges from snails and infects vertebrate hosts.
chelicerae - a pair of appendages used as mouthparts in arachnids such as scorpions, spiders, and ticks.
chemoprophylaxis - the administration of a chemical to prevent the development of an infection or the progression of an infection to active disease.
commensal - living in close association with another organism.
crepuscular - the twilight periods of light at dusk and dawn.
delayed contact sensitivity - reaction of skin or other tissue that takes 24 to 48 hours to develop and involves cell mediated immunity.
diapause - a period of arrested development and reduced metabolic rate, during which growth and metamorphosis cease.
diurnal - activities occurring during the daytime.
ectoparasite - a parasite that lives on the exterior of its host.
endemic - the constant presence of a disease or infection within a given geographic area.
endophagic - an arthropod that prefers to feed indoors.
endophilic - the tendency of arthropods to enter human structures.
enzootic - a disease that primarily infects animals and is present in an animal community at all times.
epidemic - the occurrence of cases of an illness (or an outbreak) that is clearly in excess of normal expectancy.
epizootic - an outbreak of a disease within an animal population.
eutrophic - rich in nutrients; usually applied to aquatic ecosystems.
exophagic - the tendency of an arthropod to feed outdoors.
exophilic - the tendency of blood-sucking arthropods to feed and rest outdoors.
facultative - not obligatory; characterized by the ability to adjust to circumstances.
family - a group of related genera.
focus (pl. foci) - a specific localized area.
genus (pl. genera) - a group of closely related species.
gonotrophic cycle - the time between feeding, egg development and oviposition.
immediate contact sensitivity - reaction of skin or other tissue within minutes after the interaction of a chemical antigen with antibody.
inapparent infection - the presence of infection in a host without clinical symptoms.
incidence - the number of new cases of a specific disease occurring during a certain period of time.
incubation period - the time interval between initial contact with an infectious agent and the first appearance of symptoms associated with the infection.
indigenous - living or occurring naturally in a particular environment or area.

infection rate - the proportion (expressed as a percent) of a vector or host population that is infected.
infective - an organism that can transmit an infectious agent to another individual.
instar - an insect between successive molts.
larva (pl. larvae) - the immature stage, between the egg and pupa of an insect, or the six-legged immature stage of a tick.
larvicide - insecticides used to kill larvae or immature stages of an insect.
larviparous - insects that deposit larvae rather than eggs on a host, food source, or other substrate.
maggot - legless larva of flies (Diptera).
mechanical transmission - the vector transmits the pathogen on contaminated mouthparts, legs, or other body parts, or by passage through the digestive tract without change.
miracidium (pl. miracidia) - ciliated, first larval stage in the life cycle of *Schistosoma* that penetrates and infects a snail, undergoing further development in the snail.
molluscicide - a chemical substance used for the destruction of snails and other molluscs.
myiasis - the invasion of human tissues by fly larvae.
night soil - human excrement used as fertilizer.
nosocomial - originating in a hospital or medical treatment facility.
nulliparous - a female arthropod that has not laid eggs.
nymph - an immature stage of an insect that does not have a pupal stage or an eight-legged immature tick or mite.
obligate - necessary or compulsory; characterized by the ability to survive only in a particular environment.
pandemic - a widespread epidemic disease distributed throughout a region or continent.
parous - a female arthropod that has laid eggs.
pedipalps - the second pair of appendages of an arachnid.
periurban - relating to an area immediately surrounding a city or town.
prevalence - the total number of cases of a disease in existence at a certain time in a designated area.
photoallergy - an increased reactivity of the skin to ultraviolet and/or visible radiation on an immunological basis.
photoxicity - an increased reactivity of the skin to ultraviolet and/or visible radiation on a nonimmunological basis.
pupa (pl. pupae) - a nonfeeding and usually inactive stage between the larval and adult stage.
quest (questing) - the behavior of ticks waiting in search of a passing host.
refractory - a host or vector that will not permit development or transmission of a pathogen.
reservoir - any animal, plant or substance in which an infectious agent survives and multiplies.
rodenticide - a chemical substance used to kill rodents, generally through ingestion.
ruminants - relating to a group of even-toed mammals such as sheep, goats and camels that chew the cud and have a complex stomach.
sequelae - any aftereffects of disease.
species complex - a group of closely related species, the taxonomic relationships of which are sometimes unclear, making individual species identification difficult.
steppe - a vast, arid and treeless tract found in southeastern Europe or Asia.
sylvatic - related to a woodland or jungle habitat.
synanthropic - animals that live in close association with man.
synergist - a chemical that may have little or no toxicity in itself but, when combined with a pesticide, greatly increases the pesticide's effectiveness.
transovarial transmission - passage of a pathogen through the ovary to the next generation .
transstadial transmission - passage of a pathogen from one stage of development to another after molting.
ultra low volume (ULV) - the mechanical dispersal of concentrated insecticides in aerosols of extremely small droplets that drift with air currents.
urticaria - a reaction of the skin marked by the appearance of smooth, slightly elevated

patches (wheals) that are redder or paler than the surrounding skin and often associated with severe itching.

vector - an organism that transmits a pathogen from one host to another.

vector competence - the relative capability of a vector to permit the development, multiplication and transmission of a pathogen.

vesicant - a blistering agent.

viremia - a virus that is present in the blood.

virulence - the degree of pathogenicity of an infectious agent.

wadi - a valley or bed of a stream in regions of southwest Asia and northern Africa that is dry except during the rainy season.

xerophilic - tolerant of dry environments.

zoonosis - an infectious disease of animals transmissible under natural conditions from nonhumans to humans.

zoophilic - the preference of arthropods to feed on animals other than humans.

Appendix I. Internet Websites on Medical Entomology and Vector-borne Diseases

A. Primary Sites

1. The Armed Forces Pest Management Board's website provides information about the Board as well as Army, Navy and Air Force entomology programs. Users can download Board publications, including Technical Information Memorandums, Disease Vector Ecology Profiles, and Technical Information Bulletins, and search the Defense Pest Management Information Analysis Center's literature database.
http://www.afpmb.org/

2. Emerging diseases website, with current information on disease outbreaks.
http://www.fas.org/promed

3. Iowa State University's comprehensive site on medical entomology, with excellent information on links to over 20 additional sites. http://www.ent.iastate.edu/

4. WHO disease outbreak information – emerging and communicable disease information from the WHO and its databases. The tropical medicine databases are the most useful for vector-borne diseases. Access can also be obtained to the Weekly Epidemiological Record. http://www.who.int/emc/index.html

5. The Walter Reed Biosystematic Unit's online information regarding taxonomic keys, diseases transmitted by mosquitoes, and mosquito identification modules. http://wrbu.si.edu/

6. Centers for Disease Control – information on the CDC's travel alerts, including access to country health profiles, vaccine recommendations, State Department entry requirements, and publications.
http://www.cdc.gov/travel/

7. The National Library of Medicine's biomedical databases, especially Medline. Provides complete references and abstracts to more than 9 million journal articles from biomedical publications.
http://www.nlm.nih.gov/

8. The Malaria Foundation International's site for general resources on malaria available through the worldwide web. Includes references, malaria advisories, and lists of other malaria websites.
http://www.malaria.org

9. The WHO site for information on vector-borne diseases, including disease distribution, information on disease outbreaks, travel alerts, WHO research programs, and progress on control.
http://www.who.ch/

10. The CDC's site on information available on encephalitis, as published in the Morbidity and Mortality Weekly Report. Includes case definition and disease outbreak information.
http://www.cdc.gov/epo/mmwr/other/case_def/enceph.html

11. Information from the University of Florida's website on mosquitoes and other biting flies.
http://hammock.ifas.ufl.edu/text/ig/8804.html

12. Information on ticks and other ectoparasites from the University of Rhode Island's Tick Research Laboratory. http://www.uri.edu/artsci/zool/ticklab/

13. Information on plague available from the CDC's Morbidity and Mortality Weekly Report.
http://www.cdc.gov/epo/mmwr/other/case_def/plague.html

14. A list of websites and servers pertaining to entomology from Colorado State University. Over 30 websites are listed. http://www.colostate.edu/Depts/Entomology/ent.html

B. Additional Sites

1. Lyme Disease Network – information on Lyme disease, including research abstracts, treatments for Lyme disease, newsletter, conferences, and professional resources. http://www.lymenet.org

2. The USDA plant database – includes the integrated taxonomic information system. http://plants.usda.gov/

3. University of Sydney, Medical Entomology – contains information on mosquito keys, fact sheets, and photos of mosquitoes. http://medent.usyd.edu.au

4. American Society of Tropical Medicine and Hygiene – information on the ASTMH's programs, conferences, newsletters, publications, and resources. http://www.astmh.org

5. The American Mosquito Control Association's site containing information on mosquito biology, AMCA programs, conferences, newsletters, publications, and resources. http://www.mosquito.org

6. Reuters search engine on health news pertaining to health issues around the world. http://www.reutershealth.com/

7. The ORSTOM home page includes information about the organization's medical research program in Asia, Africa, and Latin America. Bulletins and publications on its research are offered. http://www.orstom.fr/

8. Emory University's website allows access to the University's extensive database of medical and scientific literature. http://www.medweb.emory.edu/medweb/

9. The Entomological Society of America offers information on its overall services, including conferences, journals, references, membership, and literature available for ordering. http://www.entsoc.org

10. Travel Health Online contains country profiles with health precautions and disease risk summaries, general travel health advice, contacts for providers of pretravel health services, and access to US State Department publications. http://www.tripprep.com

11. The CIWEC Clinic Travel Medicine Center is the best source of western medical care in Nepal. The clinic was establish in 1982 and has focused on the health problems of foreigners in Nepal. The clinic maintains a web site that is updated regularly to provide the latest information regarding health risks in Nepal. http://ciwec-clinic.com

12. Major Scott Stockwell, U.S. Army, has compiled this website on scorpion stings, phylogeny, classification and identification as well as links to other scorpion websites. http://wrbu.si.edu/www/stockwell/classification/classification.html

Appendix J. Metric Conversion Table.

Metric System		U.S. Customary System	
LINEAR MEASURE		LINEAR MEASURE	
10 millimeters	= 1 centimeter	12 inches	= 1 foot
10 centimeters	= 1 decimeter	3 feet	= 1 yard
10 decimeters	= 1 meter	5 ½ yards	= 1 rod
10 meters	= 1 decameter	40 rods	= 1 furlong
10 decameters	= 1 hectometer	8 furlongs	= 1 mile
10 hectometers	= 1 kilometer	3 land miles	= 1 league
AREA MEASURE		AREA MEASURE	
100 sq. millimeters	= 1 sq. centimeter	144 sq. inches	= 1 sq. foot
10,000 sq. centimeters	= 1 sq. meter	9 sq. feet	= 1 sq. yard
1,000,000 sq. millimeters	= 1 sq. meter	30 ¼ sq. yards	= 1 sq. rod
100 sq. meters	= 1 sq. are	160 sq. rods	= 1 acre
100 acres	= 1 hectare	640 acres	= 1 sq. mile
100 hectares	= 1 sq. kilometer	1 sq. mile	= 1 section
1,000,000 sq. meters	= 1 sq. kilometer	36 sections	= 1 township
VOLUME MEASURE		LIQUID MEASURE	
1 liter	= 0.001 cubic meters	4 gills (2 cups)	= 1 pint
10 milliliters	= 1 centiliter	2 pints	= 1 quart
10 centiliters	= 1 deciliter	4 quarts	= 1 gallon
10 decaliters	= 1 liter		
10 liters	= 1 decaliters	DRY MEASURE	
10 decaliters	= 1 hectoliter	2 pints	= 1 pint
10 hectoliters	= 1 kiloliter	8 quarts	= 1 peck
		4 pecks	= 1 bushel
WEIGHT		WEIGHT	
10 milligrams	= 1 centigram	27 11/32 grains	= 1 dram
10 centigrams	= 1 decigram	16 drams	= 1 ounce
10 decigrams	= 1 gram	16 ounces	= 1 pound
10 grams	= 1 decagram	100 pounds	= 1 hundredweight
10 decagrams	= 1 hectogram	20 hundredweight	= 1 ton
10 hectograms	= 1 kilogram		
1,000 kilograms	= 1 metric ton		

Kitchen Measurements

3 tsp.	=	1 tbsp.	5 1/3 tbsp.	=	1/3 cup	2 cups	=	1 pint	2 pints	=	1 quart
4 tbsp.	=	¼ cup	16 tbsp.	=	1 cup	4 cups	=	1 quart	4 quarts	=	1 gallon

Temperature

Celsius = $\frac{5}{9}(F-32)$ Fahrenheit = $\frac{9C}{5} + 32$

Conversion Table

To Convert	Into	Multiply by	To Convert	Into	Multiply by	To Convert	Into	Multiply by
Centimeters	Inches	.394	Liters	Cups	4.226	Miles	Feet	5,280
	Feet	.0328		Pints	2.113		Yards	1,760
	Meters	.01		Gallons	.264		Kilometers	1.609
	Millimeters	10		Milliliters	1000	Pints	Liters	.473
				Quarts	1.057		Quarts	.5
Meters	Centimeters	100	Grams	Ounces	.035		Gallons	.125
	Feet	3.281		Pounds	.002	Quarts	Pints	2
	Inches	39.37		Kilograms	.001		Liters	.946
	Kilometers	.001	Kilograms	Grams	1,000		Gallons	.25
	Miles	.0006214		Ounces	35.274	Gallons	Pints	8
	Millimeters	1000		Pounds	2.205		Liters	3.785
	Yards	1.093	Inches	Centimeters	2.54		Quart	4
				Feet	.0833	Ounces	Grams	28.35
Kilometers	Feet	3281		Meters	.0264		Pounds	.0625
	Meters	1000		Yards	.0278		Kilograms	.028
	Miles	.621	Yards	Inches	36	Pounds	Grams	453.59
	Yards	1093		Feet	3		Ounces	16
				Meters	.914		Kilograms	.454
				Miles	.0005682			